

**DEPRESSION, ANXIETY AND QUALITY OF LIFE IN PATIENTS
WITH PSORIASIS : A CROSS SECTIONAL STUDY**

Dissertation submitted for partial fulfillment

of the rules and regulations

DOCTOR OF MEDICINE

BRANCH - XVIII (PSYCHIATRY)



THE TAMILNADU DR.MGR MEDICAL UNIVERSITY,

CHENNAI,TAMIL NADU

MAY 2019

CERTIFICATE

This is to certify that the dissertation titled, “ **DEPRESSION , ANXIETY AND QUALITY OF LIFE IN PATIENTS WITH PSORIASIS : A CROSS SECTIONAL STUDY**” is the bonafide work of **Dr. ROSE MONICA .V .R** , in part fulfillment of the requirements for the M.D. Branch – XVIII (Psychiatry) examination of The Tamilnadu **Dr. M. G. R. Medical University**, to be held in May 2019. The period of study was from Feb 2017 – Aug 2017

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CERTIFICATE OF GUIDE

This is to certify that the dissertation titled, “ **DEPRESSION, ANXIETY AND QUALITY OF LIFE IN PATIENTS WITH PSORIASIS : A CROSS SECTIONAL STUDY**” is the original work of **Dr. ROSE MONICA .V. R** , done under my guidance submitted in partial fulfillment of the requirements for M.D. Branch – XVIII [Psychiatry] examination of The Tamilnadu Dr. M. G. R. Medical University, to be held in May 2019.

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DECLARATION

I Dr.ROSE MONICA .V .R, solemnly declare that the dissertation titled,
**“DEPRESSION , ANXIETY AND QUALITY OF LIFE IN PATIENTS
WITH PSORIASIS : A CROSS SECTIONAL STUDY”** is a bonafide work
done by myself at the Madras Medical College, Chennai, during the period from
February 2017 – August 2017 under the guidance and supervision of. **Dr.**
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The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University
towards part fulfillment for M.D. Branch XVIII (Psychiatry) examination.

Place

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Dear Dr.Rosemonica V.R.,

The Institutional Ethics Committee has considered your request and approved your study titled **"DEPRESSION, ANXIETY AND QUALITY OF LIFE IN PATIENTS WITH PSORIASIS: A CROSS SECTIONAL STUDY " AT DEPARTMENT OF DERMATOLOGY, MADRAS MEDICAL COLLEGE & RGGGH, CHENNAI - NO.15022017**

The following members of Ethics Committee were present in the meeting hold on **07.02.2017** conducted at Madras Medical College, Chennai 3

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We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

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INTRODUCTION

Psoriasis is a common dermatological condition affecting skin, joints, nails and other systems. It is an autoimmune disorder where both environmental factors and genetic factors play an important role. The word PSORIASIS is derived from the Greek word 'psora' – which means itch.

Psoriasis is a psychocutaneous disorder in which there is complex interaction between skin and mind. Brain, nerve and Skin are embryological derivative of the ectoderm, and to be more precise, it is derived from the neural plate. This explains the connection between mind and skin. (1)In psoriasis, the proliferation of keratinocytes are ten times more than our normal skin. Normally, skin takes 28 to 30 days to mature and shed. But in psoriasis, keratinocytes raise to the surface in 3-6 days forming a scaly whitish patches. Since the fast dividing keratinocytes fail to mature properly, it results in raised, inflamed reddish lesions known as plaques. These plaques are very painful and itchy.

Patients with chronic illness suffer more from major depressive disorder or subsyndromal depression, which is hardly recognised. When depression is not addressed, it leads to poor quality of life and affects the individual's skin condition too, leading to severe exacerbation of skin lesion. (2, 3) Approximately 30 – 40% of patients seeking medical treatment for their skin conditions, often have an underlying psychological problem or psychiatric illness that causes or exacerbates the skin disorder. (4) In psoriasis, the

prevalence of depression and anxiety is significantly much higher than that compared to the general population. (5) We might attribute the association between mood disorders and psoriasis to shame, social anxiety or skin lesion related embarrassment. But literature shows that the prevalence of mood symptoms are higher than that is observed with many other skin disorders that are disfiguring (6- 10)

SCOPE OF THE STUDY

As many western and Indian studies show that patients with psoriasis may have depression and anxiety, some are treated but most of them go unnoticed. Literature states 5 to 10 % of patients with psoriasis commit suicide (Madhulika A gupta et al). The various psychological factors that trigger psoriasis, factors leading to symptom exacerbation and poor quality of life need to be assessed and addressed. Psychiatric morbidity has a greater influence on individual's life, So we undertook this study to determine the prevalence of depression and anxiety and its severity. An attempt was made to study the association between psychological variables (depression and anxiety) with quality of life and the severity of psoriasis.

REVIEW OF LITERATURE

The review of literature will be dealt with under five headings,

- TYPES OF PSORIASIS
- CAUSES OF PSORIASIS
- PSORIASIS AND MOOD DISORDERS
- QUALITY OF LIFE IN PSORIASIS
- SEVERITY OF PSORIASIS AND PSYCHIATRIC MORBIDITY.

Classifying psoriasis:

Clinically psoriasis manifests as well defined erythematous plaques, silvery scales, with raised and irregular borders, affecting both the upper and lower extremities and the trunk. It has a predilection for scalp, elbows and the knees. Psoriasis has significant systemic involvement too. (11, 12)

No one classification satisfies all the previous mentioned requirements. So the criteria is usually intermingled and subclasses are not exclusive. (13)

The spectrum of clinical varieties of psoriasis

- ❖ Psoriasis vulgaris
- ❖ Inverse psoriasis
- ❖ Guttate psoriasis
- ❖ Pustular psoriasis
- ❖ Palmoplantar psoriasis.

- ❖ Erythrodermic psoriasis
- ❖ Nail psoriasis

1. Psoriasis vulgaris

It is the commonest type of psoriasis, occurring in 90% of all cases. The plaques are well-delineated from the surrounding skin. Plaques are well circumscribed, elevated, superficial, solid lesion, greater than 1cm in diameter. They are Reddish or salmon pink in colour. The lesions are covered by white or silvery scales, which looks thick, thin, large or small (Figure 1).

Longitudinal studies have demonstrated that these plaques are dynamic [14] with an active, expanding edge. These lesions expand in an annular fashion, (Figure. 2) leaving clinically normal skin in the centre of the original plaque. Edge of the lesions are active, rapidly progressing lesions that are annular, with normal skin at the centre. Plaques are distributed symmetrically, and commonly present on the extensor aspects of elbows, knees, scalp, lumbosacral region, and umbilicus. Active inflammatory psoriasis has Koebner phenomenon. (15)

Koebner phenomenon also known as the koebner response or isomorphic response, is the appearance of skin lesions on the lines of skin trauma.



Figure 1. Plaque of Psoriasis Vulgaris

Henseler and Christopher have identified two ages of onset:

- ❖ TYPE I - accounting for approximately 75% of patients. occurs at or before the age of 40 years and
- ❖ TYPE II occurring at 55–60 years (16)



Figure 2. Annular psoriasis with clearance at the centre of plaque.

Site-specific variants of Psoriasis Vulgaris (PV)

A. Flexural/ intertriginous :

These are Inverse psoriasis. It is located on the skin folds - under the breasts, skin folds around the groin, armpits and between the buttocks. These lesions are subjected to irritation from rubbing and sweating because of its location over the skin folds and tender areas (Figure 3).

The major characteristic of inverse psoriasis are

- Sharply demarcated
- Erythematous plaques lesions
- With itch or burn

Plaques are very thin, have minimal scale with a shiny (nonscaly) surface with secondary fissuring or maceration. [17]. Lesions are commonly found in inguinal, intergluteal, submammary, genital folds, umbilicus. Popliteus and axilla are rarely involved. Certain contributing factors to the development of psoriasis in accordance with the Koebner phenomenon are humidity and heat typical of these sites, local traumatic factors infections caused by dermatophytes and *Candida albicans* (18)



Figure 3. Flexural psoriasis

B. Seborrhoeic:

Seborrhoeic psoriasis ('sebopsoriasis'), are so called because of its morphology and anatomical similarity to seborrhoeic dermatitis. It occurs either in isolation or in association with plaque psoriasis. Sites involved are the nasolabial folds (Figure 4), medial cheeks, nose, ears, eyebrows, hair line, scalp, presternal and interscapular regions. Characteristically these lesions are red, thin, well-demarcated. Lesions have variable degrees of scaling.



Figure 4. Seborrhoeic psoriasis, nasolabial,

C. Scalp:

The most commonest anatomical site to be involved and most frequent site of initial presentation is the scalp. Lesions range from discrete plaques to total scalp involvement with either thick plaques or scaly areas. Lesions are often asymmetrical. Sites of predilection are the postauricular area and the occiput. Plaques rarely extend > 2 cm beyond the hairline, which is the most characteristic feature.



Figure 5. Psoriasis of the scalp.

D. Palms/soles (nonpustular):

Palmoplantar pustulosis, consists of yellow-brown, sterile pustules located on the palms and soles. Around 25% of individuals with palmoplantar pustulosis have chronic plaque psoriasis. Predominantly women are found to be affected with female: male ratio of 9:1. It starts in the 4th or 5th decades of life (Figure 6) almost 95% are associated with smoking [15].



Figure 6. Psoriasis of Soles

2. GUTTATE PSORIASIS

Guttate means "drop" in Latin; it is also termed Psoriasis Exanthematic, Raindrop Psoriasis, Teardrop Psoriasis. It is the second most common type of psoriasis. It is characterized by the sudden onset of widely dispersed small red scaly plaques, presenting commonly over the trunk and the proximal limbs. Guttate psoriasis most frequently occurs in adolescents and young adults. Guttate flares in patients with psoriasis vulgaris. There are numerous red, small drop-like spots covering a large portion of the skin. These Spots have abundant scaling over them.

Site of occurrence are over the trunk, arms, legs and scalp. These lesions can eventually clear up without treatment and can resurface in the form of plaque psoriasis. GP is often associated with a preceding streptococcal throat infection or a rise in anti-streptococcal serum titer [19]. Bacterial streptococcal infections (streptococcal throat infection, chronic tonsillitis) or a viral upper respiratory tract infections infection usually precede and trigger the first signs of Guttate Psoriasis in genetically predisposed individuals.

3. PUSTULAR PSORIASIS

Generalized pustular psoriasis:

These patients develop it after pustular episodes or may have pre-existing plaque psoriasis. Acute episodes occurs with topical therapy or abrupt withdrawal of steroids (20). During an acute attack of GPP (von Zumbusch type) the skin becomes tender and very red. There may be associated fever, anorexia and nausea. Within hours, pinhead-sized pustules appear, on the erythematous background. GPP should be differentiated from acute generalized exanthematic pustulosis, a self-limiting febrile drug reaction which usually resolves within 2 weeks after withdrawal of the drug. This is an extremely rare type with high mortality rate.

In Localized pustular psoriasis, it involves :

- **Acrodermatitis continua** is a rare, chronic, pustular eruption of the toes and fingers (Figure 7). It is also known as dermatitis repens. It begins after a localized trauma starting at the tip of a single digit [21].
- **Palmoplantar pustulosis:** It is characterized by hyperkeratosis and pustules over the palmar aspects of the hands. Affects ages between 20 to 60. Eventually these pustules turn brown and peel off.



Figure 7. Acrodermatitis Continua

4. ERYTHRODERMIC PSORIASIS

Due to unidentifiable triggering factors, plaque psoriasis transitions into an inflammatory phase with predominant erythema, limited scaling, itching and rapidly progressing lesions. Erythrodermic psoriasis causes severe illness by

disrupting body's chemical balance. This is unstable and sometimes causes whole-body involvement. The erythrodermic phase has loss of peculiar clinical features of psoriasis. (Figure8).



Figure 8. Erythrodermic Psoriasis

5. NAIL PSORIASIS

Nail psoriasis occurs in 50% of patients with psoriasis. They develop characteristic nail changes as a clinical correlate of psoriatic inflammation of the nail matrix and/or nail bed. The important signs of nail psoriasis are distal onycholysis and pitting [22]. Also presents with yellowish discoloration, subungual hyperkeratosis, paronychia, onycholysis and severe onychodystrophy (Figure 9) [23].



Figure 9. Nail Psoriasis

EPIDEMIOLOGY

The prevalence of psoriasis varies worldwide. Around 2% of the population in USA are affected. Higher rates have been reported in Faroe islands, and one of the study reports reveal 2.8% of the population being affected. (24) “The prevalence of psoriasis is low in Certain ethnic groups like the Japanese, and may be absent in aboriginal Australians (25) and Indians from South America”. (26)

Presentation of psoriasis can be at any age. The age of onset of psoriasis is difficult to determine since studies rely on patient’s recall of the onset of skin lesions or physician’s diagnosis as recorded. Data based on first visit to a physician would possibly underestimate the disease onset, as skin lesions would have been present for many years before the consultation. Large studies show bimodal age of onset. At the first presentation, mean age of onset would be from 15 - 20 years of age, with second peak presenting at 55–60 years.

“In addition, strong associations have been reported with human leucocyte antigen (HLA)-Cw6 in patients with early onset, compared with later onset of psoriasis”. Psoriasis has unpredictable course and progression.

Most of the prevalence studies from India are hospital based studies, in which they have collected a comprehensive data from various medical colleges located in Dibrugarh, Calcutta, Patna, Darbhanga, Lucknow, New Delhi and Amritsar. (27) Among total skin patients, incidence of psoriasis ranges between 0.44 and 2.2%, and overall incidence being 1.02%. it was found that incidence in Amritsar (2.2%) was higher, compared to other areas in Eastern India. So they hypothesised that this may be due to genetic factors, environmental conditions (temperature) and dietary habits. (Okhandiar *et al.*) Highest incidence of psoriasis was noted among age groups 20 to 39 years. Another study done in North India by Bedi et al, reported 0.8% prevalence of psoriasis. But the limitation was the small sample size. Affected male to female sex ratio was 2.5:1 This study, showed that mean age of onset for females were low compared to males. “In the same study done by Bedi, [28] which included larger number of around 530 subjects, the prevalence of psoriasis among dermatology outpatients was found to be 2.8% while male to female ratio continued to be the same”.

A study conducted in North India, among dermatology outpatients, prevalence of psoriasis was 2.3% [29] out of which, 33% were women and 67% were men, male to female ratio was 2.03:1. Ages of these patients ranged from infancy to 8th decade and the mean age was 33.6 years. Out of this, 4.4%

patients were children. So from the above mentioned studies it could be concluded that prevalence of Psoriasis in India varies from 0.44 - 2.8%. Presenting most commonly at their third or fourth decade. Males being commonly affected than the females.. In these studies, the major limitations are the absence of validated diagnostic criteria, and time trends of disease.

2. CAUSES

A. GENETIC

Population studies reveals that the incidence of psoriasis is much greater among first-degree relatives and second-degree relatives than among general population. This is further supported by twins studies which shows, the risk of psoriasis is two to three times higher in monozygotic twins than dizygotic twins. (30) Psoriasis has a complex mode of inheritance. Genome wide linkage studies have identified around nine chromosomal loci which are statistically significant linkages; and these loci are known as “psoriasis susceptibility 1 through 9 (*PSORS1* through *PSORS9*)”. (31) Around 35 to 50% of patients have genetic determinant *PSORS1*, (32)

The heritability of the disease, and the initial finding has been replicated in multiple genome wide association studies. *PSORS1* is located within the major histocompatibility complex (MHC) on chromosome 6p, spanning an approximate 220-kb segment within the class I telomeric region of HLA-B. Three genes within this region have been the major focus of investigation because of the strong association of polymorphic coding-sequence variants

with psoriasis vulgaris. *HLA-C* (associated variant, HLA-Cw6) encodes a class I MHC protein. *CCHCR1* (associated variant, WWCC) encodes the coiled-coil, α -helical rod protein 1, a ubiquitously expressed protein that is overexpressed in psoriatic epidermis. (33)

Corneodesmosin (*CDSN*) (associated variant, allele 5) encodes corneodesmosin, a protein that is uniquely expressed in the granular and cornified layers of the epidermis. This is up-regulated specifically in psoriasis. (34). Absolute identification of the causative gene at this locus has been challenging because of the extensive linkage disequilibrium (i.e., genes on one chromosome are inherited together and are not easily separable by recombination events) observed within the MHC. Current data suggest that HLA-Cw6 is the susceptibility allele within *PSORS* (35, 36); However, no disease-specific mutations have been identified so far, and the variants in regulatory sequences are potentially affecting several other downstream genes.. *HLA-C* is an interesting candidate gene, Studies show that it might be involved in immune responses at the levels of both antigen presentation and natural killer–cell regulation.

Studies have clearly shown that the clinical variants of psoriasis are genetically heterogeneous at least at the level of *PSORS1*. Thus, guttate psoriasis, an acute-onset form usually occurring in adolescents, is strongly associated with *PSORS1* (37) whereas late-onset cases of psoriasis vulgaris (cases in persons over 50 years of age) and palmoplantar pustulosis are not associated with *PSORS1*. (38) The implications of genetic heterogeneity for

disease management have yet to be determined, but such heterogeneity, clearly points on to the potentially distinctive disease entities beyond the descriptive pattern of illness.

There is only very little data on genetic studies in India. A study which included 67 psoriasis patients from Western India found an association with A1, B17 and Cw6 antigens, but did not with B13 (Chablani *et al.*)[39]

Another study conducted in south india found association of HLA Bw57 and DR7 (Pitchappan *et al.*) (40)

In North India, Cw*0602 was the major allele which presented in higher frequency among the psoriasis patients (Rani *et al.*) (41)

Genetic predispositions of the disease was greater in childhood psoriasis than adult onset psoriasis. (42-44) Indian studies have reported lower familial incidence of psoriasis. A positive family history of psoriasis was found only in 14% of patients (45). While Kaur *et al.* had reported family history in only 2% of subjects. In 84% of the cases, majority of first degree relatives were found to be affected and 12% cases were second degree relatives. (46) Only few studies have reported familial inheritance of the disease, statistically significant data on familial incidence are not available.

B. Psoriasis and psychological distress

Aristotle emphasised a link between physical health and mood. This has drawn the attention of many investigator's interest to establish a link between mental disorders and psoriasis. Psychodermatology is a new discipline in psychosomatic medicine which studies the influence of stress in exacerbation and chronicity of the disorder. It emphasises the fact that both are interconnected at embryonal level through the ectoderm.

A study done by Kimball et al showed impairment in psychological functioning in psoriasis patients, by psychological impairment they refer to "cumulative life course impairment" (Kimball et al). (47). According to this model, psychological burden due to psoriasis negatively influences many aspects of patient's life like relationships, occupation, social interaction and wellbeing. The concept of "cumulative life course impairment" entails the effect of stigma, psychological and medical co-morbidities. Psoriasis has economic as well as social consequences that ultimately results in failure to achieve "full life potential" in most of the patients. (47, 48). Patients with chronic illness have reported that psoriasis has a negative influence on relationships, job decisions, educational status, self-esteem and well-being (48)

A chronic illness is said to influence the environmental, Psychological and social aspects of a patient's life and the illness progression has overall impact on the life conditions of patient. Patients with severe psoriasis presenting with multiple site involvement and greater progression of disease,

presented with a greater psychological distress, low autonomy, and low purposes in life. They had also reported feelings of demoralization. Demoralization is a condition with feelings of helplessness and a belief that they have failed to meet one's expectations and goals. Demoralized individuals have difficulty coping with problems and stressful life events (49) Picardi et al reported that patients with severe psoriasis also reported higher rates of Type A behaviour (50). Type A behaviour is characterized by individuals who are competitive and overcommitted to work, when these individuals are subjected to deadlines or time pressure they tend to experience a sense of pervasive urgency (51).

Apart from this psychological distress also plays a significant role in onset, maintenance and exacerbation of psoriasis. Psychological distress also influences the treatment adherence. It was found that 37 – 71% of patients with psoriasis had psychological distress. So it could be concluded that Genetic, personality, social and emotional factors are found to perpetuate the illness. Previous researcher have found out that stress appraisal and reactivity were mostly associated with younger age, poor quality of life, greater severity of the illness and more psoriasis related stress. Almost 50% of the patients with psoriasis had their onset related to a stressful life event (charlotte ramrod et al) It is also interesting to note that association between onset and stress was associated with depression and anxiety

A study by sreelatha lakshmy et al, conducted in southern india, included 90 patients, among whom 51 patients had significant stress with associated psychiatric morbidity and poor quality of life.

Another study demonstrated, that chronic psychological stress will alter the epidermal barrier functions. Following repeated stripping of skin with cellophane tape, causing disruption of integrity, there was prolongation of recovery of epidermal permeability barrier functions. This extent of prolongation was positively correlated with patient's intensity of stress (52).

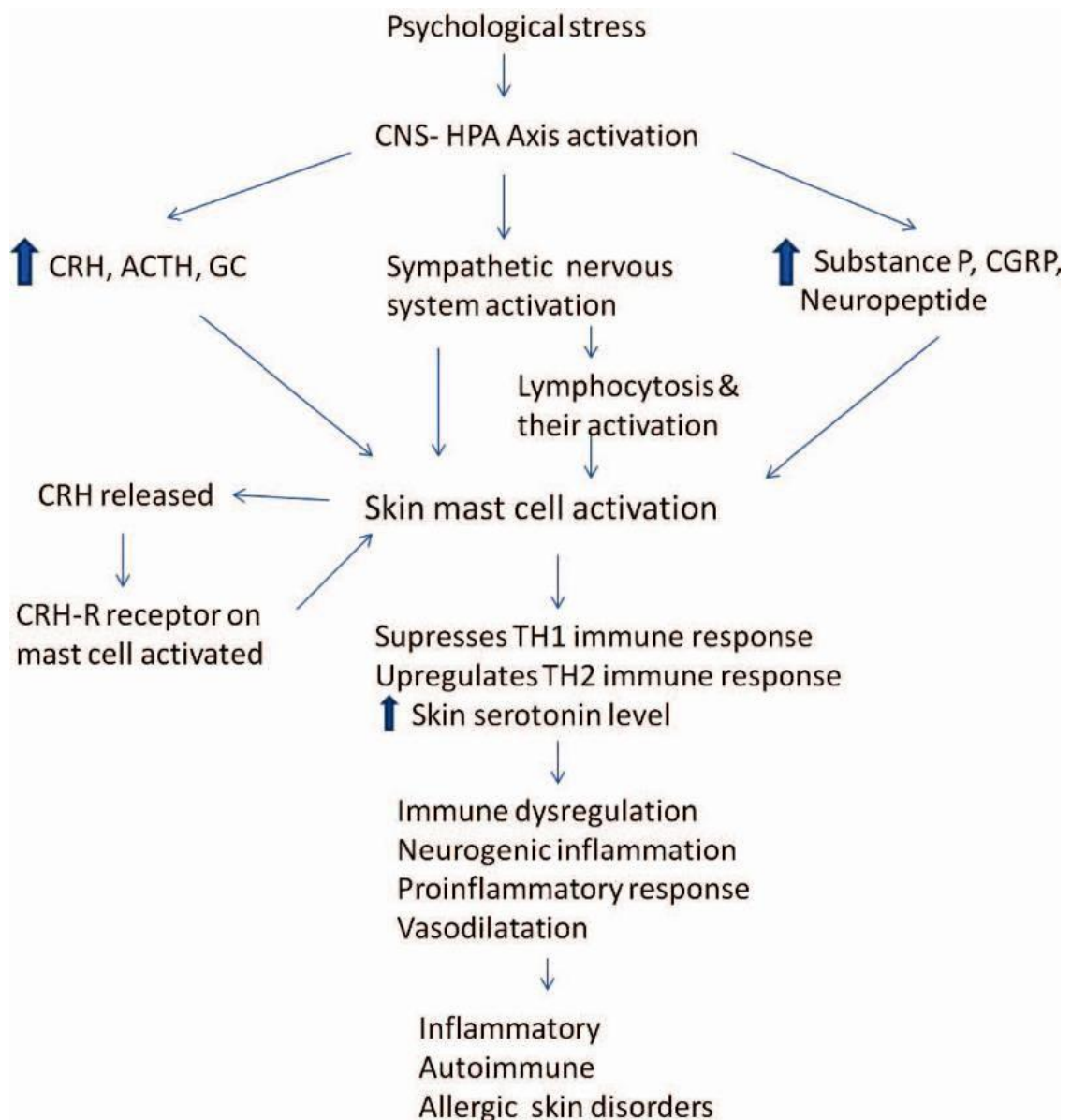


Figure.10. Interplay of various factors causing skin disorders from psychological stress (Savita yadav et al 2013)

3. PSORIASIS AND MOOD DISORDERS

In 1990's, a concept of Disability adjusted life years (DALY) was developed in order to compare the overall health and the life expectancy of an individual. In 1996, world health organisation adopted this method. DALY is a measure which denotes the burden of a disease. It is expressed in number of years lost due the illness or disability or early death. Mental disorders and neurological disorders accounts for around 10% of the total disability adjusted life years. Research shows consistent evidence that people with progressive or recurrent medical problems are affected with mental health problems (53)

Many studies have shown that there is a physiological connection between psoriasis and mood disorders. Inflammatory mediators have been implicated in the pathophysiology of both psoriasis and depression. We will see in detail the pathophysiology of psoriasis and mood disorders.

In major depressive disorder, there are elevated levels of proinflammatory cytokines namely prostaglandins (PGE₂), C-reactive protein (CRP) and Interleukins like IL-2, IL-6, IL-1 β and TNF- α . It also shows a dose-response relationship with severity of Depressive disorder (54, 55). However all these findings, cannot differentiate whether depression is due to inflammation or vice versa. Studies exploring the temporal association between the two, have found evidences and biological plausibility for occurrence in both directions.

Important factors that play a major role,

1. Depression causing inflammatory states.
2. Hypothalamo-pituitary- adrenal axis
3. HPA Axis Effects on Skin and Inflammation
4. Role of melatonin.

A. Depression Causing Inflammatory states-.

Can depression result in elevated levels of inflammatory cytokines ?

Yes, Randomized controlled trials in depressed subjects have revealed that, treatment with antidepressants like sertraline, a selective serotonin reuptake inhibitor (SSRI), have shown a significant decrease in depressive symptoms, and also decrease in the level of inflammatory mediators like of CRP and IL-6 (56). A large metaanalysis by Steptoe et al, describes how psychological stress elevates proinflammatory markers namely CRP, IL-6, TNF- α and IL-1 (57). So the overall effect of depression was an acute increase in proinflammatory cytokines. In the context of psoriasis, this would mean, that stress increases the inflammatory cytokines and thereby causing an exacerbation of the condition. In around 40–80% of individuals with psoriasis had their onset or exacerbation of lesions due to psychosocial factors (58).

B. Hypothalamic-pituitary- adrenal axis

Hypothalamic-pituitary- adrenal axis is one of the central stress response system in our body. It mediates the neuroendocrine adaptation to a stressful situation. This response is characterised by the release of corticotrophin

releasing hormone (CRH) or corticotrophin releasing factor (CRF) from the hypothalamus. CRH binds to CRF receptors in the anterior pituitary gland and releases adrenocorticotrophic hormone (ACTH). ACTH binds on to adrenal receptors in the adrenal gland and stimulates the release of cortisol. In the view of homeostasis, excessive release of cortisol is controlled by the negative feedback mechanism, where the excessive cortisol is looped back to hypothalamus and stops the further release of ACTH.

Cortisol effects are mediated by two distinct intracellular receptors namely, mineralocorticoid receptors (MR, type I) and glucocorticoid receptors (GR, type II) (59). Mineralocorticoid receptors have greater affinity for endogenous glucocorticoids. Whereas glucocorticoid receptors show more affinity for synthetic steroids like dexamethasone. With these profiles, mineralocorticoid receptors are postulated to modulate the cortisol effects and its feedback on the HPA axis whenever the cortisol levels were low.

When an individual confronts with a stressful situation, our body exerts a heightened cortisol response, and with repeated exposure to stress, the individual learns to habituate to the stressful situation by persistent repeated HPA axis activation. This persistent activation leads to chronically elevated levels of CRH, ACTH and Cortisol levels. HPA axis hyperactivity is possibly attributed to the change in the number and function of these cortisol receptors. This mechanism is supported by the dexamethasone suppression test, where there is non suppression of cortisol levels following an administration of

dexamethasone. This effect is found to disappear after clinical recovery from depression (60). Hypercortisolemia causes negative effects on individual's mood and cognition. These effects are thought to be mediated through, mineralocorticoid receptors or possibly through glucocorticoid receptors, especially those which have not lost the functional capacity as the ones in hypothalamus, that participate in HPA autoregulation.

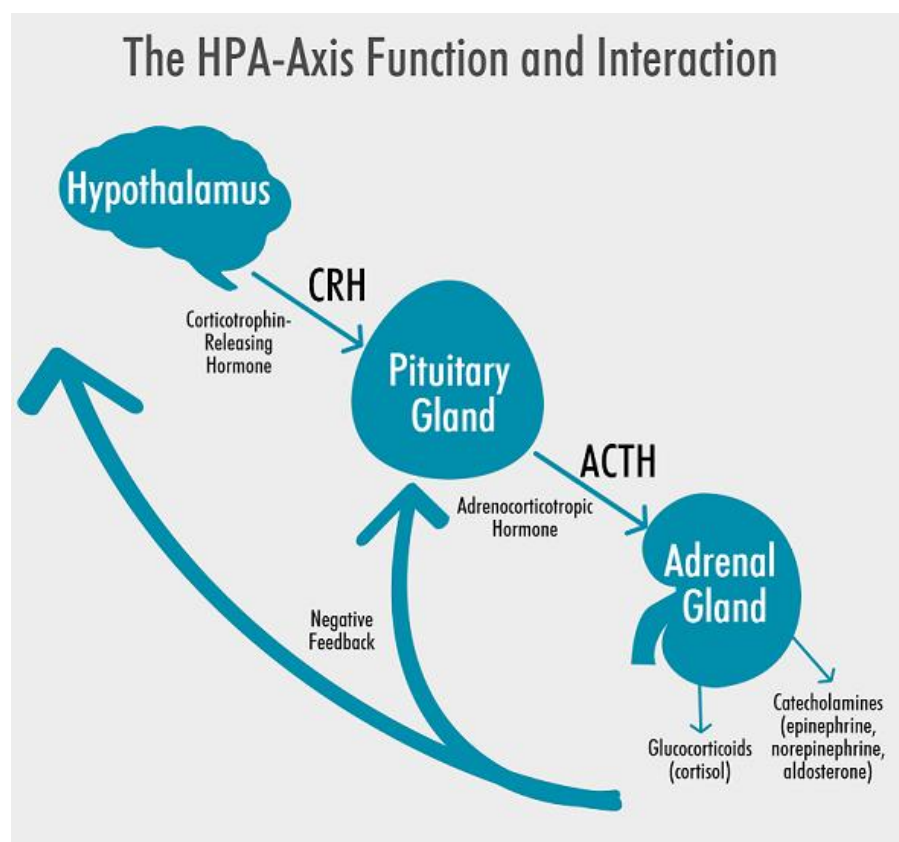


Figure 11. Hypothalamic - pituitary - adrenal axis

C. HPA Axis Effects on Skin and Inflammation.

Apart from the mood changes, HPA hyperactivity also exerts its effects on skin. CRH plays a very vital role in the interaction between central and peripheral stress response. CRH has been demonstrated to stimulate the local

cytokine production in the skin (cytokines namely IL-6 and IL-11) (61). CRH also amplifies cytokine-mediated keratinocyte expression in the immune trafficking adhesion molecules like hCAM and ICAM-1, and the major histocompatibility complex II receptor, HLA-DR (62).

“In addition to this, CRH activates the proinflammatory protein complex NF- κ B (nuclear factor kappa-light-chain-enhancer of activated B cells), which are present in almost all cell types and modulates DNA transcription and immune reaction in response to stimuli like stress, UV light, free radicals, and bacterial/viral infection” (63). With all of these changes, CRH significantly shifts the keratinocytes into an immunoactive state, thereby contributing to inflammatory skin conditions, like psoriasis. Psoriatic lesions when biopsied and analysed, showed significant increase in the expression of CRH as compared to that of normal skin, suggesting a possibility of role of CRH in pathophysiological mechanism of psoriasis (64).

Depending on whether the patient suffers from depression or anxiety, HPA axis is said to be altered. Patients suffering from depression have elevated levels of cortisol, CRH and ACTH. These patients also have a blunted HPA-stress response, which means less cortisol is released following an acute stress (65).

Depression and Anxiety worsens psoriasis and the severity of lesions. Even though these are mediated through separate mechanisms, In acute anxiety state, inflammatory cytokines are released in excess, without appropriate

production of anti-inflammatory cortisol. So due to lack of cortisol production, it doesn't seem to mitigate the cutaneous response in psoriasis.

Chronic stress in the form of anxiety is found to delay the wound healing. Anxiety sensitises the patient for trauma induced psoriatic lesions and also causes a delay in healing of the lesions. (66) Apart from the HPA axis contributions for the causation of psoriasis, the sympathetic nervous system (SNS) is also implicated in the pathophysiology. In Anxiety, there is excessive sympathetic activation (67). Similarly, patients with depression also demonstrate an increased sympathetic tone. Studies also demonstrate an increased muscle sympathetic nervous system activity (MSNA) found at rest and during psychological stress.

This increased MSNA effect is more pronounced when depression score increases and it reduces on treatment with SSRI sertraline (68). Patients with major depression, have higher levels of plasma norepinephrine, which is an indirect measure of SNS activity. This sympathetic overactivity is found to normalise on treatment with antidepressants SSRIs (69) and desipramine (70). The sympathetic nervous system innervates lymphoid organs, thereby promoting inflammation (71) Studies demonstrate sympathetic-mediated upregulation of proinflammatory cytokines namely IL-6 and IL-1 β (72). Among the mediators of inflammation, norepinephrine in particular is a major effector of sympathetic enhancement of immune reactivity (73).

Saint-Mezard et al. investigated the role of psychological stress in psoriasis and the inflammatory response. He found that there was an augmented dendritic cell response with consequent increase in T-cell activation. In order to find whether this effect was mediated by norepinephrine or due to stress induced glucocorticoid release, he investigated selective depletion of NE, which effectively normalized the dendritic cell migratory activity and emerging hypersensitivity reaction. Patients with psoriasis secrete excessive amounts of NE in response to an acute stress when compared to controls. So he concluded that, when compared to controls, individuals with psoriasis had sympathetic overactivity that contributed to an inflammatory state and lead to the exacerbation of psoriasis (74)

Sympathetic stress response is mediated mainly by, the alpha adrenergic receptors (α -ARs). These receptors are expected to cause an increased cytokine production (75). The Norepinephrine which is released in response to stress, causes an activation of α -ARs located in the macrophages and dendritic cells. This subsequently leads to an increase in the release of TNF- α and suppression of anti-inflammatory mediator IL-10. This effect will lead to an exacerbation of the underlying inflammatory state. In contradiction to this, activation of β 2-ARs causes suppression of TNF- α release and increases IL-10 and IL-2 production.

Due to opposing actions of these two adrenergic receptor subclasses, immunological stress reaction of an individual is consistently dependent on the varied expression of α - and β -ARs within that individual and the balance of

their effects. In chronic inflammatory Conditions like rheumatoid arthritis, when treated with β 2-agonists, it did not elicit any change in cytokine production, but treatment with an α -antagonist altered the cytokine profile and modulated the immune functions (Lubahn et al.).Thereby it was concluded that chronic inflammatory conditions would alter the of sympathetic nervous system functions and, this in turn regulates the immune activity through these receptor classes. So, in psoriasis also, literature shows the use of β -blockers in these patients have reported to cause an exacerbation of lesions and also triggers the onset of psoriasis (76, 77)

Studies have also revealed that TNF- α and IL-10, being key components of inflammatory pathway influences the fibroblast growth factor10 (FGF 10). FGF 10 causes keratinocyte proliferation and thereby causing psoriasis lesions. (78, 79) This was further confirmed by immunostaining method which revealed increased expression of FGF 10 in psoriatic lesions. On further investigating the influence of FGF 10 on psoriasis, a study done by yao et al found, when neutralizing the FGF 10 with monoclonal antibody, there was improvement in skin lesions in guinea pigs. (47)

- ❖ So from these studies it can be concluded that acute stress triggering SNS activity, causes release of nor epinephrine that disproportionately activates inflammatory α -adrenergic receptors that leads to worsening of disease by means of various chemical messengers like TNF- α and FGF10.

Some had hypothesized that immunoreactivity following an acute stress has an evolutionary adaptation, this adjustment is basically to prepare the body to defend against Infections that occur following an injury (80, 81).

So based on each individual's personal experience, coping skills, cognition, the subjective interpretation of a stressor varies accordingly. Research done by Carroll et al showed, tasks related increase in anxiety and anger are positively correlated with higher serum levels of inflammatory mediator IL-6. This again demonstrates that the emotional responses are instrumental in determining the subsequent immune reactivity (82). This finding has been replicated in many other studies. In addition, others have discovered that good social support, decreased subjective stress experience and IL-6 levels (83).

Depression per say will influence psoriasis and the disease severity. Patients with depression will have cognitive distortions, that alters the way they perceive their environment. (84, 85). So depressed subjects have decrease in perceived degree of social support, a parameter that improves with treatment of depression. Investigators also suggests that under acute stress, the increase in stress immune reactivity, in patients with low social support, might be an evolutionary adaptation that occurs to stress. Under such circumstances, stress had only amplified the disease process and worsen the psoriasis severity rather than increasing the defence against infections as evolutionary adaptation.

Depression and anxiety is predominantly influenced by chronic stress, rather than acute stress. What could be the consequences of chronic stress, and how it alters the course of inflammatory disease is very interesting. Evidence suggests that chronic stress results in immunosuppressed state or dysregulation of immune system. As previously discussed, in depression, chronic stress causes HPA axis hyperactivity with elevated levels of ACTH, CRH, and cortisol. Also, in acute stress, HPA axis response is blunted, which means, there is no increase in cortisol levels, and diurnal cycle of cortisol secretion is also disrupted (86).

These changes are correlated with the deleterious effects of chronic stress on depression — manifested as decreased wound healing, further aggravating the skin lesions by increased production of inflammatory cytokines. This is also emphasised to be the primary means through which stress induces or exacerbates autoimmune disorders. Considering all this, evidences show that depression and anxiety also contribute to worsening of inflammatory diseases especially psoriasis, through HPA axis and SNS hyperactivity. Furthermore, individuals with mood disorders demonstrate disruptions in their physiological systems which results in progression of immunopathology. Additionally, these individuals have increased responsiveness to acute stressors, leading to altered immune function and causing acute worsening of chronic inflammatory and autoimmune disorders.

Recent investigations by Akayo et al showed there is a link between psoriasis and depression (87), data of Kirby et al revealed that patients suffering from psoriasis (88) had higher incidence of depression and anxiety. Another study by Mazetti et al also establish higher prevalence of mental disorders among psoriasis patients, 71.2% patients with psoriasis had psychiatric diagnosis, among which 35% had personality disorders, 17.5% had depression, 12.5% had anxiety disorders and 6.25% with schizophrenic trait.

A similar study done by Darko Biljan, which included 70 participants with mean age of 51 years, showed 90% of the participants had psychiatric diagnosis. Among which 19.2% had affective disorders, 13.7% had mixed anxiety and depression, 16.4% had alcohol use disorder. This study also found that 80% of participants had stressful life events before the onset of psoriasis. Comparing with Indian studies, it also shows that psychiatric morbidity is more common in patients with psoriasis, the prevalence being slightly less than western studies- 53.3%. The prevalence of depression was reported to be 23.3% and anxiety was 3.3% (89).

Another study conducted at Mumbai, followed up patients with psoriasis and examined for psychiatric morbidity. The Study showed these patients had adjustment disorder in 62%, depressive episode in 29% and dysthymia in 4% (90). Studies also show that alcohol use and smoking is also commonly associated with psoriasis. (91, 92)

It is also interesting to note that, among many other chronic dermatological disorders, psoriasis was significantly associated with psychiatric morbidity and the prevalence of psychiatric illness was also increased in psoriasis compared to other skin disorders. Reviewing the Indian studies, study by Sagar B. Karia et al, compared the psychiatric morbidity in psoriasis and alopecia areata. He included 50 participants with psoriasis and 50 participants with alopecia areata. The study reported psychiatric morbidity of 22% in alopecia areata and 38% in psoriasis. Among this depression was diagnosed in 18 % and anxiety in 4% of alopecia areata. 24% depression and 12% anxiety in psoriasis.

The study also reports other psychiatric profile of 65% having adjustment disorders of depressed type, 30% depressive episodes, and 4% dysthymia (93).

A cross sectional analysis of prevalence of depression and anxiety conducted at Mahatma Gandhi Medical College and Research Institute, Puducherry investigated 90 patients with psoriasis. The results showed, prevalence of depression was 78.9% and the prevalence of anxiety was 76.7%, which was significantly more than the previously mentioned studies. (94)

C. ROLE OF MELATONIN

Apart from inflammatory mediators, melatonin also plays a very important role in linking depression and psoriasis. There are disruptions in the secretion of melatonin in depression (95) Melatonin secretion is regulated by

the circadian rhythm. Elevated levels are found at night, peaking in the early morning between 2am to 4 a.m. (96). Melatonin is a hormone, that not only regulates the daily sleep cycle, but also modulates the immune functions.

By reducing levels of inflammatory cytokines like TNF- α , IL-6, and IL-8, melatonin could theoretically attenuate the severity of inflammatory disorders (97, 98) Melatonin dysregulation has been studied in many other inflammatory disorders, including sarcoidosis, psoriasis vulgaris (99, 100)

These disruptions in cyclical secretion of melatonin could contribute to inflammatory states.. Night time melatonin levels in patients with psoriasis has been significantly lower than in healthy controls. Decreased melatonin levels would lead to previously mentioned Koebner phenomenon. This is responsible for exacerbation of the skin lesions in psoriasis. This was further confirmed by delayed wound healing in rats that underwent pinealectomy, which has caused a depletion in melatonin and further with melatonin replacement, this effect was eradicated (101).

Melatonin secreting hormone (MSH) levels may be elevated in depression due to low melatonin levels and hypersecretion of CRH. This contributes to the clinical presentation of psoriasis. Interestingly, many other studies have implicated MSH as an indirect contributor to depressive symptoms (102, 103). This was proved by the improvement in depressive symptoms following administration of MSH inhibitor has (104)

Low melatonin levels are also associated with morbidity in psoriasis. A prospective cohort study done with 14, 128 participants demonstrated an increase in the risk of development of diabetes mellitus type II (DMII) in psoriasis patients than the control group. The risk was directly proportional to the severity of the psoriasis (105).

This implies that melatonin regulates the blood glucose levels through action on melatonin receptors present in the pancreatic beta cells and on the insulin receptors in the pituitary. (106)

The interrelationship between diabetes and melatonin has been demonstrated in studies where an increase in the levels of insulin was accompanied by decreased.

Melatonin levels, decreased pineal insulin receptors, and increased pancreatic melatonin receptors

Apart from depression and diabetes mellitus, psoriasis is associated with other conditions with chronic inflammatory pathophysiology like – myocardial infarction, systemic hypertension, stroke, and results in increased mortality. Melatonin may directly influence these medical co morbidities by its anti-inflammatory properties. Melatonin administration has reported to decrease blood pressure in untreated, hypertensive patients. (cavallo et al 2004, E.Grossman et al, 2006).

In addition, Melatonin also protects against endothelial dysfunction and ischemic heart disease (A. O.Sehiril et al, 2013) This further emphasises the

potential of widespread clinical use of melatonin, in at-risk psoriatic population.

Phototherapy is one of the treatment modalities used for treating depression. Phototherapy is also used in the treatment for psoriatic lesions. The mechanism underlying this in psoriasis is not known, but its conceptualized as due to the inhibition of keratinocyte proliferation, and immunomodulation (107- 109).

Considering sunlight as the main zeitgeber for regulating the circadian rhythm of secretion of melatonin, Melatonin may be used to study its therapeutic effect in treating both depression and psoriasis. In fact, agomelatine, a melatonin receptor agonist, has already shown its efficacy in the treatment of depression

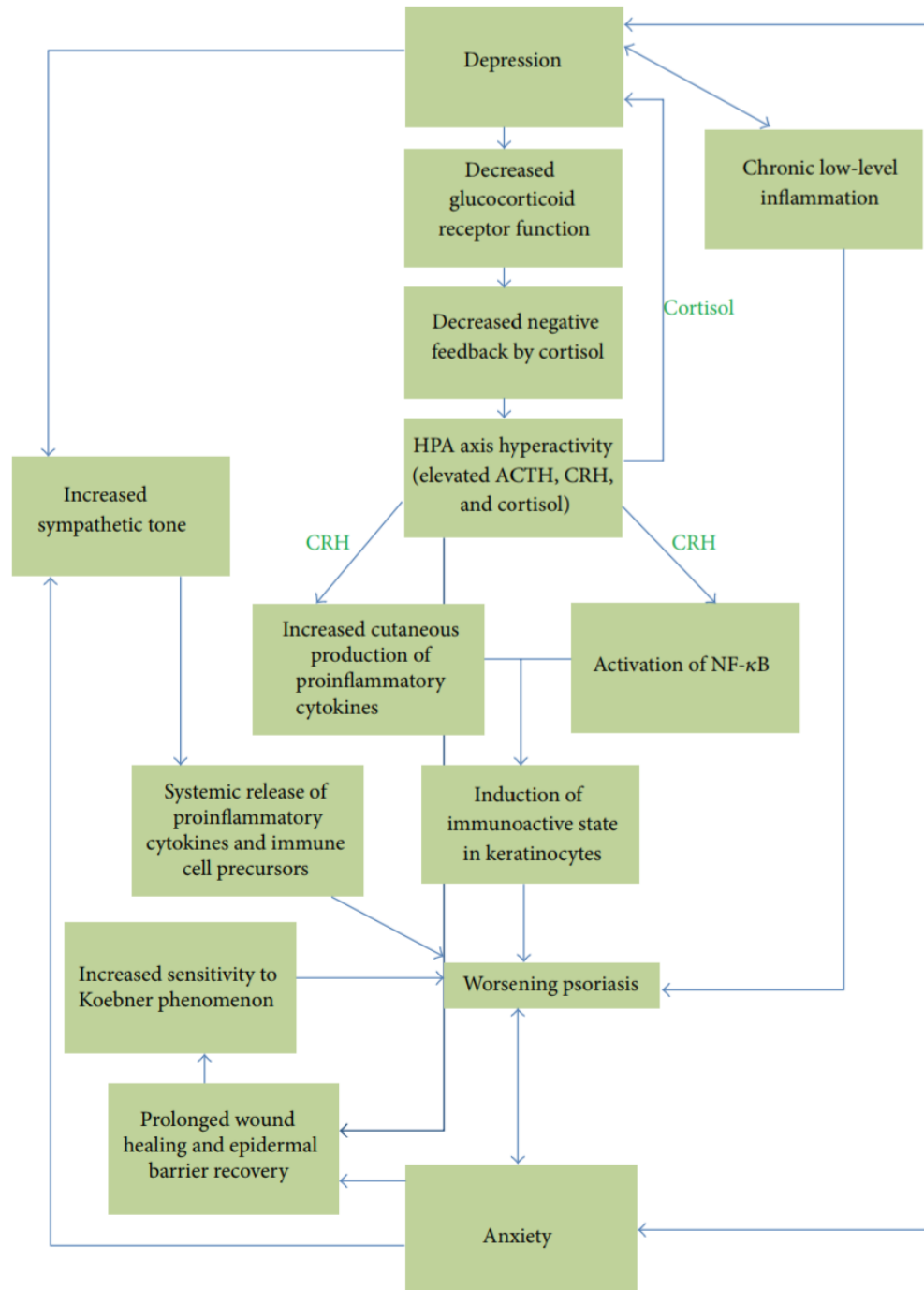


Figure. 12 Overview of mood disorders and psoriasis (Cody J et al, 2015)

5. QUALITY OF LIFE IN PSORIASIS

Skin being the largest and most visible organ in our body, to a greater extent it determines our external appearance. The way we look is very important and it influences the way how we are perceived by others. Skin plays a major role in social and sexual communication. Skin diseases cause an altered or impaired appearance and seem to profoundly influence those affected with skin diseases.

Apart from causing physical discomfort and personal inconvenience, it has been reported that they influence a person's life in terms of daily functioning and psychological status. Skin disease also provokes negative emotions like shame and embarrassment, anxiety, poor self esteem and lack of confidence. Apart from discomfort in social relationships, patients also experience inferiority, they also feel discriminated and stigmatized. Thus as a whole skin diseases significantly affect patient's quality of life (QOL) (110).

A study done in lokmanya tilak municipal medical college, Mumbai had reported quality of life scores negatively correlated with Hamilton –D, Hamilton –A scores, which means that depression and anxiety was correlated to poor quality of life. Also increased severity of psoriasis was accountable for poor quality of life. (111)

6. SEVERITY OF PSORIASIS AND PSYCHIATRIC MORBIDITY

Psoriasis being a chronic inflammatory disorder, affects not only the skin, but other systems as well. The degree of systemic involvement is determined by the severity of psoriasis. This is further supported by the evidence of increased skin messenger RNA expression of IL – 32 A, IL -17, IL- 22 in moderate to severe psoriasis. (Balato et al, 2014). Also serum levels of inflammatory cytokines, vascular endothelial growth factors, fibrinogen and C-reactive protein are positively related to the severity. (Choe et al, 2012).

A commonly held view is that, if skin inflammation is less, then severity of psoriasis is mild. This is a common misconception because, the severity is assessed only by cutaneous examination of cellular immune infiltrates.

Chronic skin lesions were mostly associated with psychiatric morbidity, which could be explained by the biological overlapping of immune pathology.

FACTORS INFLUENCING THE DISEASE SEVERITY

- i. Location of lesions
- ii. Extent of the lesions
- iii. Degree of inflammation
- iv. Response to treatment
- v. Quality of life.

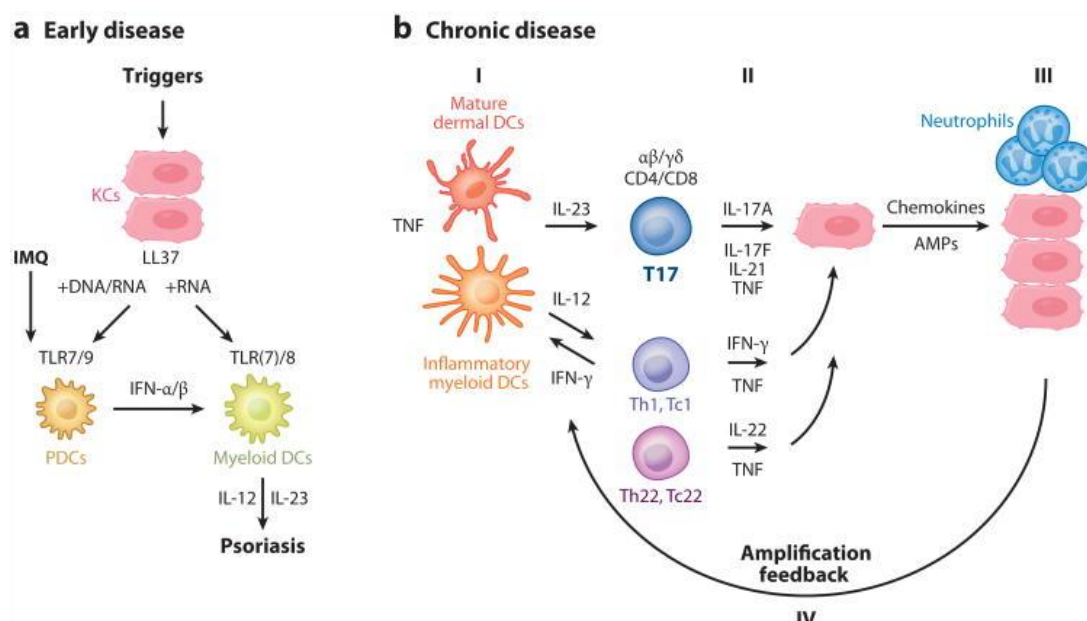


Table13 Immune reaction in acute and chronic disease

Table 13. explains the increase in immune mediators as the illness progresses. Activation of various cytokines and chemokines causes increase in the disease severity and also the role of cortisol influences the disease progression.

Severity of psoriasis is assessed by “PSORIASIS AREA AND SEVERITY INDEX” (PASI), Body surface area (BSA), Physician global assessment (PGA). PASI it is most widely used for quantifying the severity of psoriasis. It is an objective measure of clinical severity. For a quick assessment, “RULE OF TENS” is applied. It denotes severe disease if any one of the following is present, PASI score more that 10, DQLI score more than 10 or body surface area of more than 10.

Most of the studies report higher psychiatric morbidity, especially depression and anxiety in patients with many lesions and more severe disease

but on the contrary, a swedish study reports 1---Psychological morbidity was not associated with increased PASI score (112- 114)

An Indian study done at Maharashtra found 22.33 % rate of psychiatric disorders. (115). This was a cross sectional study that included 103 patients.the profile of psychiatric disorders in this study was

- 65% adjustment disorder depressed type
- 30 % depressive disorder
- 4% dysthymia
- However there was no anxiety disorders detected in this study.

AIMS AND OBJECTIVES

PRIMARY OBJECTIVE

1. To assess the prevalence and severity of depression and anxiety among patients with psoriasis.
2. To assess the quality of life among patients with psoriasis

SECONDARY OBJECTIVE

1. To check for any association between psoriasis severity with psychiatric morbidity
2. To assess the quality of life with severity of psychopathology and the severity of psoriasis.

METHODOLOGY

The study was conducted at the department of Dermatology, Madras Medical College, Chennai. The thesis abstract was presented at the Institution Ethics Committee and approval was obtained. Study was done from Feb 2017 to Aug 2017. All consecutive patients aged >18 years, diagnosed with psoriasis and undergoing treatment at department of dermatology were included in the study irrespective of their psoriasis type, severity and duration of illness. Patients were selected based on inclusion and exclusion criteria. Psoriasis area and severity index (PASI) was estimated by the dermatologist in order to rate the severity of psoriasis. Patients were explained about the study in detail and informed consent was taken from the patient and then recruited for the study.

Both inpatients and out patients were taken for the study. A total of 114 patients were screened and 100 patients fulfilling the criteria were enrolled in the study. This is a cross sectional study, conducted for 6 months from the date of ethics committee approval. Their sociodemographic data, psychological variables and other relevant details were collected using a proforma and relevant scales were applied. The collected data were analysed and necessary results were obtained.

INCLUSION CRITERIA

1. Participants diagnosed as psoriasis by dermatologists (both inpatients and outpatients)
2. age >18 years
3. Both men and women
4. Patients selected irrespective of the type of psoriasis, duration of illness, severity of the disease and treatment.

EXCLUSION CRITERIA

1. Other chronic skin diseases which impairs the quality of life
2. Physical disability
3. Chronic medical illness.
4. Psychosis, previously diagnosed mental illness
5. Cognitive impairment
6. Any Substance dependence

ASSESSMENT SCALES

1. Hamilton rating scale for depression (HAM-D)
2. Hamilton rating scale for anxiety (HAM-A)
3. Dermatology life quality index (DLQI)
4. Brief psychiatric rating scale (BPRS)

INSTRUMENTS USED

I. SOCIODEMOGRAPHIC DATA SHEET

A structured profoma was used to elicit information about the demographic details of the participants and the illness characters.

II. HAMILTON RATING SCALE FOR DEPRESSION

It is a multiple item questionnaire to assess the severity of depression. It is also helpful to assess the remission and recovery of depression. (116) MAX HAMILTON published the scale in 1960, the scale was revised in the subsequent years. The questionnaire is mainly to rate the severity of depression in adults. (117 -121)

HAM-D is an observer rating scale. It contains 21 items in total, 17 items were added to assess the severity and the rest of the 4 items for additional clinical information. Each item is scored on a 3 to 5 point scale. The total score is then compared with the descriptor to assess the severity. It takes approximately 15 – 20 minutes for assessment.

SCORING

MILD	8-13
MODERATE	14- 18
SEVERE	19-22
VERY SEVERE	>23

III. HAMILTON RATING SCALE FOR ANXIETY

HAM –A is a clinician rating questionnaire to assess the severity of anxiety. The scale was originally published in the year 1959 by Max Hamilton. (122) He developed the scale to estimate the severity of symptoms in anxiety neurosis, and not to be used as means to diagnose anxiety in other disorders. (123)The scale contains 14 items, each item contains a number of symptoms which are rated on a scale of zero to four. Upon the completion of evaluation, the clinician then sums up the 14 individually rated items and arrive at a total score. This ranges from 0 to 56.

SCORING

MILD	14 – 17
MODERATE	18 – 24
SEVERE	25 -30

IV. DERMATOLOGICAL QUALITYOF LIFE INDEX

It measures the impact of skin disease on quality of life. It was designed by Andrew Y Finlay and Gul Karim Khan in 1994. (124, 125) It is a 10 item questionnaire covering following areas – symptoms, restrictions in social and leisure activities, occupation, close relationships, sex and treatment. Each question is scored from 0 to 3.This scale is available in 115 languages (126)

NO EFFECT ON PATIENT'S LIFE	0-1
SMALL EFFECT	2-5
MODERATE EFFECT	6-10
VERY LARGE EFFECT	11-20
EXTREMELY LARGE EFFECT	21-30

V. BRIEF PSYCHIATRIC RATING SCALE

It is a rating scale used by the clinician to measure psychiatric symptoms like anxiety, depression, hallucination and unusual behaviour. (127) BPRS was published in 1962.It is the oldest and widely used scale in the field of psychiatry to measure the psychotic symptoms.There are total of 24 items, rated from 1 to 7 based on clinical evalution and also based on patient's self report.

STATISTICAL ANALYSIS

The results were tabulated and analyzed using the statistical package SPSS 22.0.

Descriptive statistics was used to obtain the mean and standard deviations with respect to different variables of socio-demographic profile and the illness characteristics of the study population. Pearson correlation was used to assess the relationship between severity of psoriasis, quality of life, depression and anxiety.

Scatter diagram was used to represent the correlation between the severity of psoriasis, quality of life, depression and anxiety.

Independent samples T-test was used to compare the mean values of age with HAM- D and HAM- A scores. Comparison of the proportion of demographic variables, psoriasis illness characteristic with depression and anxiety was done by using chi square test.

RESULTS

The study was conducted at the Department of Dermatology at Madras Medical College, Chennai. Patients who were selected are both outpatients and inpatients. Initially full physical examination was done to assess the physical status of the patient and to look for any medical co morbidity.

A total of 114 patients were enrolled, out of which 8 did not satisfy the inclusion criteria, 6 did not give their consent. So finally 100 participants were included in the study and informed consent was obtained. Study population consisted of 48 males and 52 females.

Table.1 Study Population

MALE	48
FEMALE	52
TOTAL	100

1. SOCIODEMOGRAPHIC CHARACTERISTICS

Participants had different education levels, 71% were illiterates, 22% had primary education and only 7% had completed degree.

Our study, all subjects were from lower socioeconomic background.

Majority of them worked as coolie (38%), 22% were employed, 18% were unemployed and rest of them were students, homemaker.

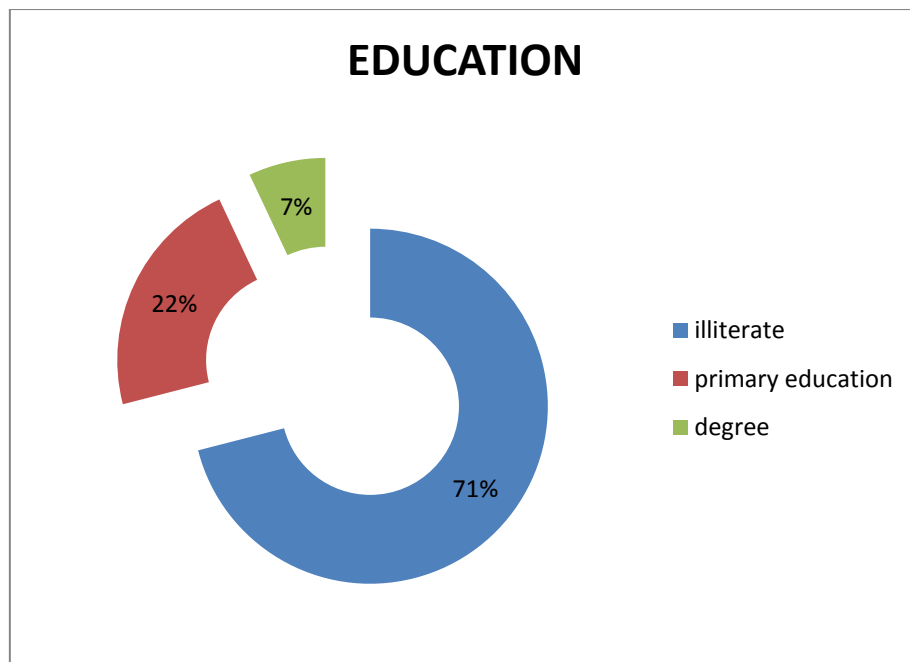


Table 2. Education of the study population

In our study, 88% were married, 9% unmarried and 3% were widow.

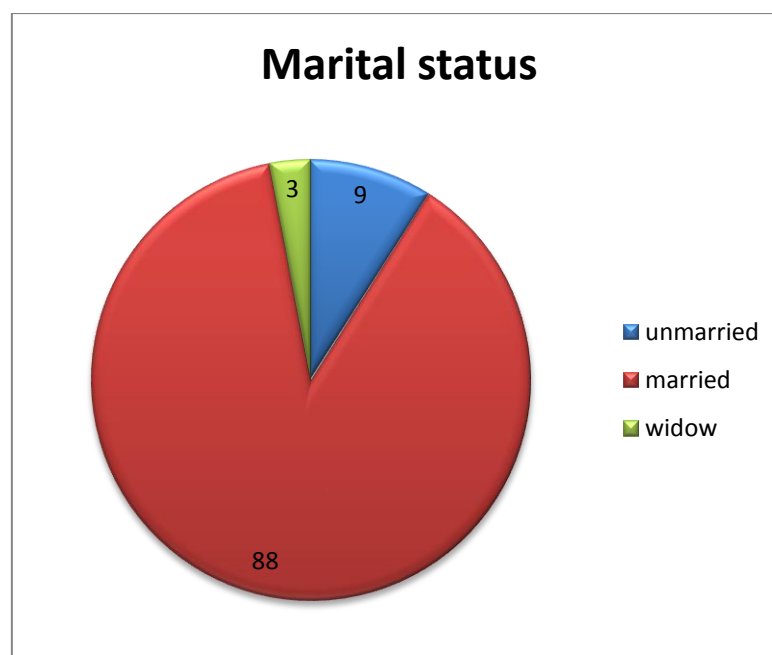


Table.3 Marital status of the study population

Most of our participants were belonging to Hinduism (77%), Christianity (14%), Islam (9%)

2. PSORIASIS ILLNESS CHARACTERISICS

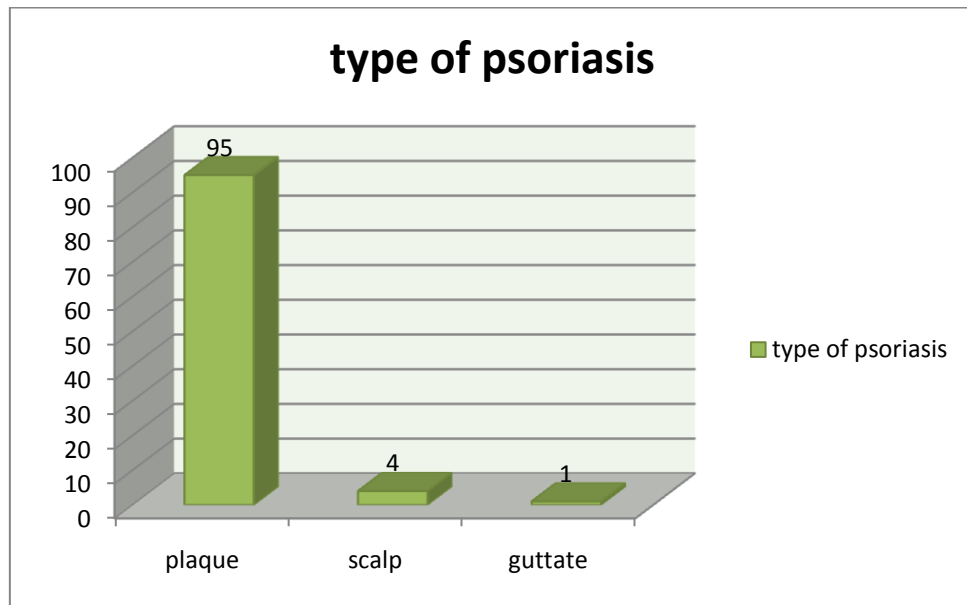


Table 4. types of psoriasis

- Out of the 100 patients, 88 were outpatients and 12 were inpatients.
- Among them 95% had plaque type psoriasis, 4% had scalp psoriasis, 1 % had guttate psoriasis.
- All of them had duration of psoriasis for more than 5 years, 2 patients with maximum of 15 years.
- Around 44 of them had more than 3 symptom exacerbation in the past 5 years.
- 8 of them were receiving topical and systemic therapy, 92 patients were on topical therapy alone.
- None had a family history of psoriasis.

- Using psoriasis area and severity index, 61% had mild severity, 25% had moderate and 14% had severe psoriasis

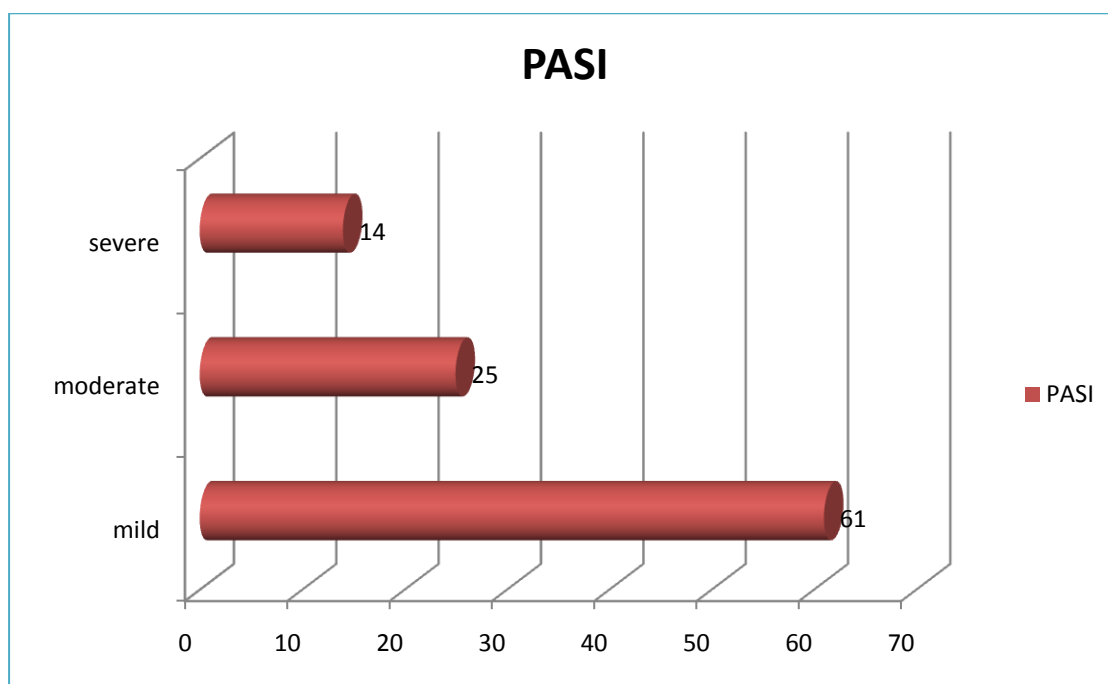


Table 5.PASI score of the study population.

3. PREVALENCE OF DEPRESSION AND ANXIETY

Among the 100 participants, 43% had significant depression and 57% did not have depression

Among them 6 % had mild depression, 20% had moderate depression, 11% had severe depression, 5% had very severe depression. Among them 4 had suicidal ideations.(table 6)



Table 6. severity of depression

Anxiety was present in 20 participants, 4 had mild anxiety, 16 had moderate anxiety. There were no patients with severe anxiety.

Assessing for psychological variables, nil had a family h/o psychiatric illness. 92% reported of good family support and 8 % had poor family support pertaining to their illness.

4. QUALITY OF LIFE

Quality of life was affected in most of the participants. extremely large effect in 15%. Very large effect in 32%, moderate effect in 37%, small effect in 16%

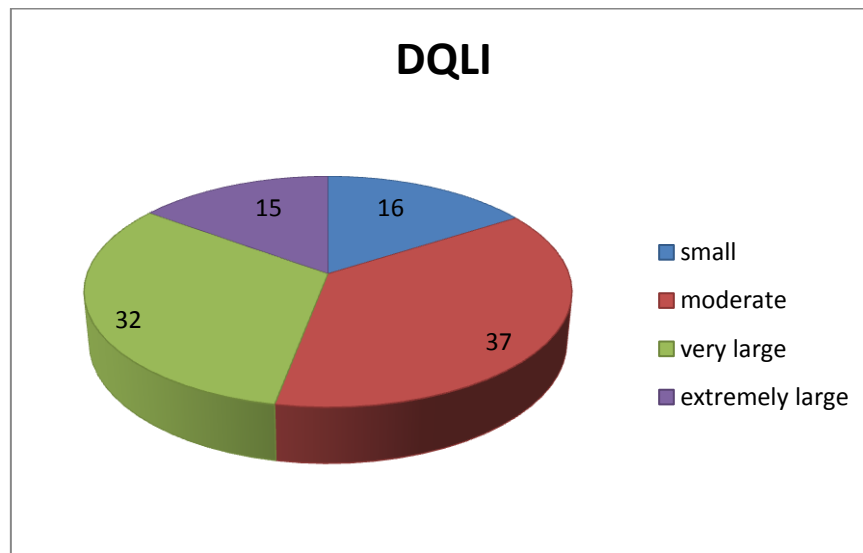


Table 7. pie chart of quality of life

Descriptive statistics

	Age (years)	PASI score	DLQI score	HAM D score	HAM A score
N	100	100	100	100	100
Mean	42.94	6.67	11.85	10.60	10.28
Std. Deviation	10.112	3.924	6.325	6.779	5.290
Median	44.00	5.00	9.50	7.00	8.00
Minimum	24	2	3	2	4
Maximum	66	21	26	26	22

Table 8. Descriptive Statistics

The mean age in the study population was 42.94 ± 10.11 , the mean score of psoriasis severity is 6.67 ± 3.924 , the mean score of DQLI was 11.85 ± 6.325 , the mean scores of HAM-D and HAM – A was 10.60 ± 6.779 , and 10.28 ± 5.290 respectively.

5. COMPARING THE ASSOCIATION OF DEMOGRAPHIC VARIABLES WITH DEPRESSION.

Most of the socio-demographic variables were insignificant with respect to depression among psoriasis patients. But depression was significantly associated with

- Type of psoriasis,
- Mode of treatment (IP/OP),
- Severity of psoriasis and
- Poor quality of life.

A. TYPE OF PSORIASIS

Psoriasis type	Depression (HAM-D)					
	No		Yes		Total	
	N	%	N	%	N	%
Plague	57	60.0%	38	40.0%	95	100.0%
Scalp	0	0.0%	4	100.0%	4	100.0%
Guttate	0	0.0%	1	100.0%	1	100.0%
Total	57	57.0%	43	43.0%	100	100.0%

Chi-Square Test	Value	df
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Fisher's Exact Test	6.520	0.013
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Table 9

There was significant association between type of psoriasis and the depression scores, it was found that, out of the 95 plaque psoriasis, 38 patients had depression. Out of 4 cases of scalp psoriasis and 1 case of guttate psoriasis, all these 5 patients had severe depression.

B. MODE OF TREATMENT

There was also significant association between depression and inpatient care of treatment. Among the 88 outpatients, 32 had depression. And among the 12 inpatients, 11 of them had clinical depression.

Out of the 12 inpatients, 8 of them were on systemic steroid therapy.

Table 10 shows the significant association of depression with inpatient patients.

Patient type	Depression (HAM-D)					
	No		Yes		Total	
	N	%	N	%	N	%
Out patient	56	63.6%	32	36.4%	88	100.0%
In-patient	1	8.3%	11	91.7%	12	100.0%
Total	57	57.0%	43	43.0%	100	100.0%

Chi-Square Test	Value	df
Pearson Chi-Square	13.177	<0.001

C. SEVERITY OF PSORIASIS

It was also noted that, Out of 61 patients who had mild severity of psoriasis, 24 patients had depression. In 25 patients with moderate severity, 7 had depression. In 14 patients with severe psoriasis, 12 had depression.

PASI severity	Depression (HAM-D)					
	No		Yes		Total	
	N	%	N	%	N	%
Mild	37	60.7%	24	39.3%	61	100.0%
Moderate	18	72.0%	7	28.0%	25	100.0%
Severe	2	14.3%	12	85.7%	14	100.0%
Total	57	57.0%	43	43.0%	100	100.0%

Chi-Square Test	Value	df
Trend Chi-Square	5.146	0.023

Table 11. Severity of psoriasis

D.QUALITY OF LIFE

The more severe the impairment in quality of life, it was associated with increase in number of depression. Patients who had moderate to extremely large effect of impairment had higher depressive scores.

DLQI effect	Depression (HAM-D)					
	No		Yes		Total	
	N	%	N	%	N	%
No effect	0	0.0%	0	0.0%	0	0.0%
Small effect	14	87.5%	2	12.5%	16	100.0%
Moderate effect	26	70.3%	11	29.7%	37	100.0%
Very large effect	16	50.0%	16	50.0%	32	100.0%
Extremely large effect	1	6.7%	14	93.3%	15	100.0%
Total	57	57.0%	43	43.0%	100	100.0%

Chi-Square Test	Value	Df
Trend Chi-Square	22.965	<0.001

Table 12 Quality of life

No significant association was found in the following variables,

Gender	Depression (HAM-D)					
	No		Yes		Total	
	N	%	N	%	N	%
Male	27	56.3%	21	43.8%	48	100.0%
Female	30	57.7%	22	42.3%	52	100.0%
Total	57	57.0%	43	43.0%	100	100.0%

Chi-Square Test	Value	df
Pearson Chi-Square	0.021	0.884

Table 13 gender and depression

Education	Depression (HAM-D)					
	No		Yes		Total	
	N	%	N	%	N	%
Nil	42	59.2%	29	40.8%	71	100.0%
Primary	11	50.0%	11	50.0%	22	100.0%
Degree	4	57.1%	3	42.9%	7	100.0%
Total	57	57.0%	43	43.0%	100	100.0%

Chi-Square Test	Value	df
Fisher's Exact Test	0.673	0.794

Table 14 education and depression

Marital status	Depression (HAM-D)					
	No		Yes		Total	
	N	%	N	%	N	%
Unmarried	4	44.4%	5	55.6%	9	100.0%
Married	52	59.1%	36	40.9%	88	100.0%
Widowed	1	33.3%	2	66.7%	3	100.0%
Total	57	57.0%	43	43.0%	100	100.0%

Chi-Square Test	Value	df
Fisher's Exact Test	1.552	0.536

Table 15 marital status and depression

Substance use	Depression (HAM-D)					
	No		Yes		Total	
	N	%	N	%	N	%
None	42	56.8%	32	43.2%	74	100.0%
Alcohol	7	50.0%	7	50.0%	14	100.0%
Nicotine	8	66.7%	4	33.3%	12	100.0%
Total	57	57.0%	43	43.0%	100	100.0%

Chi-Square Test	Value	Df
Pearson Chi-Square	0.739	0.691

Table 16. substance use and depression

Religion	Depression (HAM-D)					
	No		Yes		Total	
	N	%	N	%	N	%
Hindu	42	54.5%	35	45.5%	77	100.0%
Christian	10	71.4%	4	28.6%	14	100.0%
Muslim	5	55.6%	4	44.4%	9	100.0%
Total	57	57.0%	43	43.0%	100	100.0%

Chi-Square Test	Value	Df
Fisher's Exact Test	1.379	0.562

Table 17 religion and depression

Past h/o psy	Depression (HAM-D)					
	No		Yes		Total	
	N	%	N	%	N	%
None	57	57.6%	42	42.4%	99	100.0%
Depression	0	0.0%	1	100.0%	1	100.0%
Total	57	57.0%	43	43.0%	100	100.0%

Chi-Square Test	Value	df
Fisher's Exact Test	-	0.430

Table 18 past h/o depression and depression

Family support	Depression (HAM-D)					
	No		Yes		Total	
	N	%	N	%	N	%
Good	52	56.5%	40	43.5%	92	100.0%
Poor	5	62.5%	3	37.5%	8	100.0%
Total	57	57.0%	43	43.0%	100	100.0%

Chi-Square Test	Value	df
Fisher's Exact Test	-	0.999

Table 19 family support and depression

Occupation	Depression (HAM-D)					
	No		Yes		Total	
	N	%	N	%	N	%
Nil	6	33.3%	12	66.7%	18	100.0%
Coolie	24	63.2%	14	36.8%	38	100.0%
Housewife/ Student	12	54.5%	10	45.5%	22	100.0%
Employed	15	68.2%	7	31.8%	22	100.0%
Total	57	57.0%	43	43.0%	100	100.0%

Chi-Square Test	Value	df
Pearson Chi-Square	5.878	0.118

Table 20 occupation and depression

	Depression (HAM-D)	N	Mean	Std. Dev	t-value	p-value
Age (years)	Yes	43	42.30	11.342	0.546	0.586
	No	57	43.42	9.151		

Table 21 Independent samples T-Test to compare mean age

6.COMPARING THE ASSOCIATION OF DEMOGRAPHIC VARIABLES WITH ANXIETY

A.TYPE OF PSORIASIS : Among the 95 plaque psoriasis, 15 of them had anxiety. All patients who had scalp and guttate psoriasis had higher anxiety score.

Psoriasis type	Anxiety (HAM-A)					
	No		Yes		Total	
	N	%	N	%	N	%
Plaque	80	84.2%	15	15.8%	95	100.0%
Scalp	0	0.0%	4	100.0%	4	100.0%
Guttate	0	0.0%	1	100.0%	1	100.0%
Total	80	80.0%	20	20.0%	100	100.0%

Chi-Square Test	Value	df
Fisher's Exact Test	15.630	<0.001

Table 22comparison of psoriasis type and HAM -A

B.MODE OF TREATMENT : Again as previously mentioned, the number of patients affected with depression were significantly higher in the inpatients with psoriasis. Here also we can find that, Out of 88 outpatients, 13 had anxiety and among 12 inpatients, 7 had anxiety, which is significantly greater than the outpatient population.

Patient type	Anxiety (HAM-A)					
	No		Yes		Total	
	N	%	N	%	N	%
Out patient	75	85.2%	13	14.8%	88	100.0%
In-patient	5	41.7%	7	58.3%	12	100.0%
Total	80	80.0%	20	20.0%	100	100.0%

Chi-Square Test	Value	df
Fisher's Exact Test	-	0.002

TABLE 23comparison mode of treatment and HAM -A

C. SEVERITY OF PSORIASIS

Out of 61 patients who had mild severity of psoriasis, 9 patients had anxiety. In 25 patients with moderate severity, 1 had anxiety. In 14 patients with severe psoriasis, 10 had anxiety. So here again we can see that most of the patients with severe psoriasis were having anxiety disorder.

PASI severity	Anxiety (HAM-A)					
	No		Yes		Total	
	N	%	N	%	N	%
Mild	52	85.2%	9	14.8%	61	100.0%
Moderate	24	96.0%	1	4.0%	25	100.0%
Severe	4	28.6%	10	71.4%	14	100.0%
Total	80	80.0%	20	20.0%	100	100.0%

Chi-Square Test	Value	df
Trend Chi-Square	12.649	<0.001

Table 24 comparison of PASI score and HAM-A

D.QUALITY OF LIFE

As seen with depression, anxiety scores were higher with severe impairment in quality of life. Patients who had moderate to extremely large effect of impairment had higher anxiety scores.

DLQI effect	Anxiety (HAM-A)					
	No		Yes		Total	
	N	%	N	%	N	%
No effect	0	0.0%	0	0.0%	0	0.0%
Small effect	16	100.0%	0	0.0%	16	100.0%
Moderate effect	34	91.9%	3	8.1%	37	100.0%
Very large effect	23	71.9%	9	28.1%	32	100.0%
Extremely large effect	7	46.7%	8	53.3%	15	100.0%
Total	80	80.0%	20	20.0%	100	100.0%

Chi-Square Test	Value	df
Trend Chi-Square	17.787	<0.001

Table 25 COMPARING QUALITY OF LIFE WITH ANXIETY SCORES

7. DQLI and PASI

DLQI effect	PASI severity							
	Mild		Moderate		Severe		Total	
	N	%	N	%	N	%	N	%
Small effect	10	16.4%	6	24.0%	0	0.0%	16	16.0%
Moderate effect	24	39.3%	11	44.0%	2	14.3%	37	37.0%
Very large effect	22	36.1%	7	28.0%	3	21.4%	32	32.0%
Extremely large effect	5	8.2%	1	4.0%	9	64.3%	15	15.0%
Total	61	100.0%	25	100.0%	14	100.0%	100	100.0%

Chi-Square Test	Value	df
Fisher's Exact Test	23.383	<0.001

Table 26 comparing DQLI WITH PASI

The above table26. shows that patients with severe psoriasis had poor quality of life. out of the 14 patients with severe psoriasis, 9 had extremely large effect, 3 had very large effect i.e poor quality of life

No significant association was found in the following variables.

Gender	Anxiety (HAM-A)					
	No		Yes		Total	
	N	%	N	%	N	%
Male	37	77.1%	11	22.9%	48	100.0%
Female	43	82.7%	9	17.3%	52	100.0%
Total	80	80.0%	20	20.0%	100	100.0%

Chi-Square Test	Value	df
Pearson Chi-Square	0.491	0.484

Table 27 comparison of gender and HAM-A

Occupation	Anxiety (HAM-A)					
	No		Yes		Total	
	N	%	N	%	N	%
Nil	12	66.7%	6	33.3%	18	100.0%
Coolie	31	81.6%	7	18.4%	38	100.0%
Housewife/ Student	18	81.8%	4	18.2%	22	100.0%
Employed	19	86.4%	3	13.6%	22	100.0%
Total	80	80.0%	20	20.0%	100	100.0%

Chi-Square Test	Value	df
Fisher's Exact Test	2.506	0.499

Table 28 comparison of occupation and HAM-A

Marital status	Anxiety (HAM-A)					
	No		Yes		Total	
	N	%	N	%	N	%
Unmarried	6	66.7%	3	33.3%	9	100.0%
Married	72	81.8%	16	18.2%	88	100.0%
Widowed	2	66.7%	1	33.3%	3	100.0%
Total	80	80.0%	20	20.0%	100	100.0%

Chi-Square Test	Value	df
Fisher's Exact Test	2.196	0.346

Table 29 comparison of marital status and HAM-A

Education	Anxiety (HAM-A)					
	No		Yes		Total	
	N	%	N	%	N	%
Nil	59	83.1%	12	16.9%	71	100.0%
Primary	15	68.2%	7	31.8%	22	100.0%
Degree	6	85.7%	1	14.3%	7	100.0%
Total	80	80.0%	20	20.0%	100	100.0%

Chi-Square Test	Value	df
Fisher's Exact Test	2.410	0.275

Table 30 comparison of education and HAM-A

Religion	Anxiety (HAM-A)					
	No		Yes		Total	
	N	%	N	%	N	%
Hindu	62	80.5%	15	19.5%	77	100.0%
Christian	13	92.9%	1	7.1%	14	100.0%
Muslim	5	55.6%	4	44.4%	9	100.0%
Total	80	80.0%	20	20.0%	100	100.0%

Chi-Square Test	Value	df
Fisher's Exact Test	4.326	0.115

Table 31 comparison of religion and HAM-A

Substance use	Anxiety (HAM-A)					
	No		Yes		Total	
	N	%	N	%	N	%
None	62	83.8%	12	16.2%	74	100.0%
Alcohol	9	64.3%	5	35.7%	14	100.0%
Nicotine	9	75.0%	3	25.0%	12	100.0%
Total	80	80.0%	20	20.0%	100	100.0%

Chi-Square Test	Value	df
Fisher's Exact Test	3.198	0.200

Table 32 comparison of substance use and HAM-A

Family support	Anxiety (HAM-A)					
	No		Yes		Total	
	N	%	N	%	N	%
Good	75	81.5%	17	18.5%	92	100.0%
Poor	5	62.5%	3	37.5%	8	100.0%
Total	80	80.0%	20	20.0%	100	100.0%

Chi-Square Test	Value	df
Fisher's Exact Test	-	0.196

Table 33 comparing family support and HAM -A scores

Independent samples T-Test to compare mean age

	Anxiety (HAM-A)	N	Mean	Std. Dev	t-value	p-value
Age (years)	Yes	20	42.35	12.721	0.243	0.810
	No	80	43.09	9.440		

Table 34 Independent samples T-Test to compare mean **age**

Hence from the above findings, there was significant association of depression and anxiety with

- Type of psoriasis,
- Mode of treatment (IP/OP),
- Severity of psoriasis and
- Poor quality of life.

7.CORRELATION OF PASI WITH DEPRESSION AND ANXIETY

		HAM D score	HAM A score
PASI score	Correlation	0.339	0.497
	p-value	0.001	<0.001
	N	100	100

Table 35 Correlation between severityof psoriasis with depression and anxiety

The above table shows correlation between PASI scores with depression (HAM D) and Anxiety (HAM A). There is a significant positive correlation between psoriasis severity and psychiatric morbidity

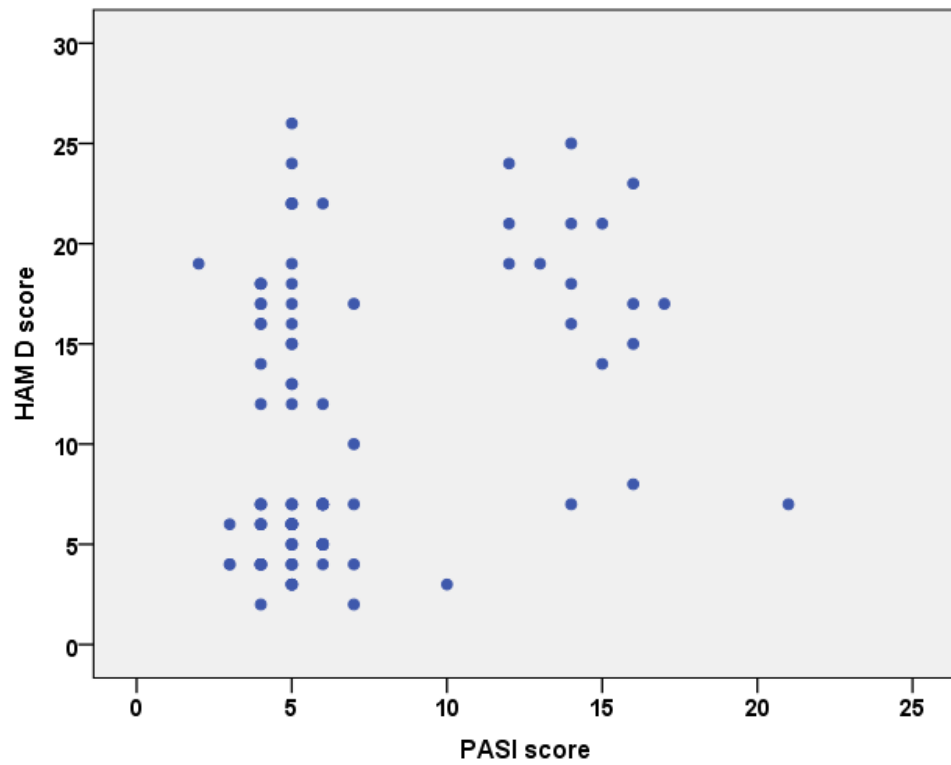


Table 36 scatter diagram showing correlation between HAM D and PASI

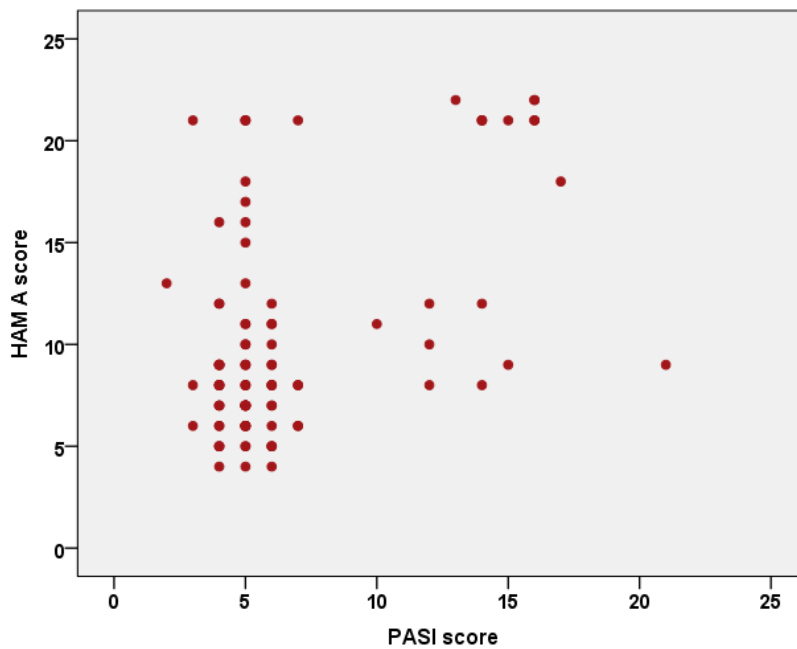


Table 37. scatter diagram showing correlation between HAM A and PASI

Correlation of quality of life with depression and anxiety

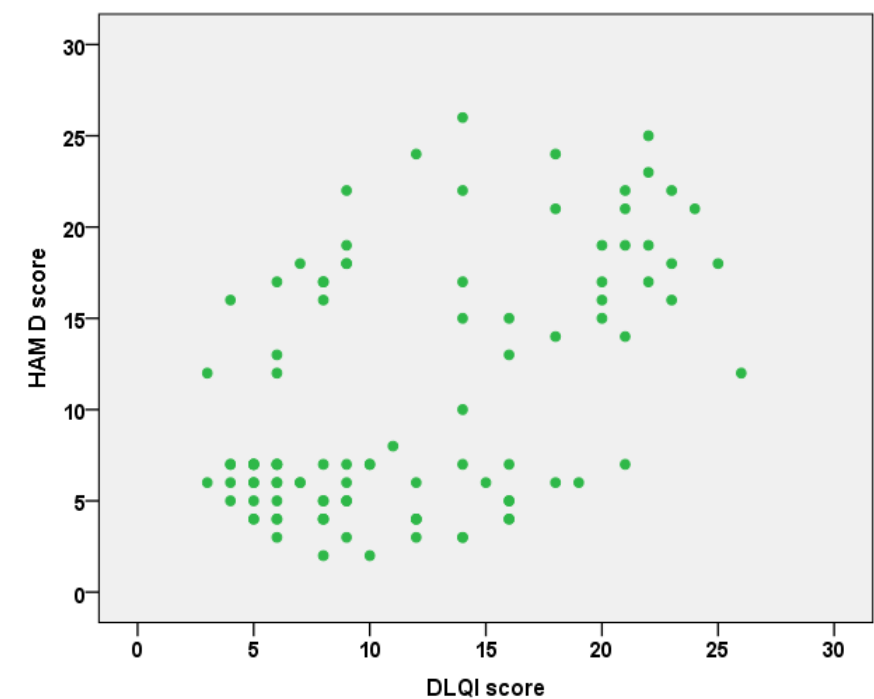


Table 38 scatter diagram showing correlation between HAM D and DQLI

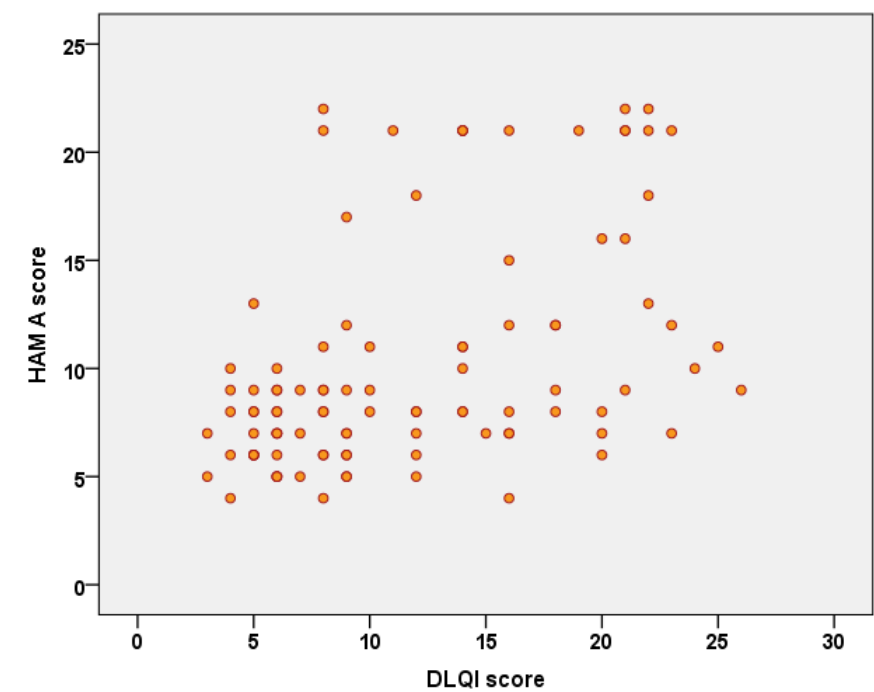


Table 39 scatter diagram showing correlation between HAM A and DQLI

9.CORRELATION OF QUALITY OF LIFE AND PSORIASIS SEVERITY

		PASI score	DLQI score	HAM D score	HAM A score
PASI score	Correlation	1	0.402	0.339	0.497
	p-value		<0.001	0.001	<0.001
	N	100	100	100	100
DLQI score	Correlation	0.402	1	0.516	0.462
	p-value	<0.001		<0.001	<0.001
	N	100	100	100	100
HAM D score	Correlation	0.339	0.516	1	0.456
	p-value	0.001	<0.001		<0.001
	N	100	100	100	100
HAM A score	Correlation	0.497	0.462	0.456	1
	p-value	<0.001	<0.001	<0.001	
	N	100	100	100	100

Table 40. correlation between severity of psoriasis, quality of life and psychiatric illness

The above table shows that quality of life and PASI scores are positively related, and also there is significant positive correlation of PASI scores with Depression and Anxiety scores.

DISCUSION

Biological interaction between psoriasis and stress may result in psychiatric disorders among psoriasis patients. Most of the psoriasis patients feel stigmatized for being assessed by a psychiatrist, hence refuse to consult, and Dermatologist also misses subtle psychiatric signs and symptoms in these patients.

Both dermatological and psychiatric consequence seems to induce, prolong and exacerbate each other. In the current study, prevalence and severity of depression was analysed among outpatients and inpatients attending tertiary care centre. We also studied the sociodemographic variables, clinical variables and their influence on mental health of patients with psoriasis.

PREVALENCE AND SEVERITY OF DEPRESSION

Our study found an overall prevalence of depression in 43 % of patients, among them 36 patients had moderate to very severe depression, that warranted psychiatric intervention.

Various studies show a prevalence ranging from 28 – 67 % (128- 132). An Indian study done by lakshmy et al reported an even much higher prevalence of 78.9% (133)

The prevalence of depression in our study is in par with previous studies and suggests that depressive symptoms are more frequent in patients with psoriasis. Our study has also found that most of the inpatients were affected, compared to the outpatients. The reason could be attributed to the severity of psoriasis requiring hospitalisation. And some patients were also on systemic steroid therapy, which could have caused steroid induced depression too. Also our study revealed that depression was significantly associated with scalp and guttate psoriasis. Again the reason could be visible site of involvement (scalp) and extent of skin lesions with pruritis. This association was not previously assessed in other studies and needs further investigation.

Although 43 % patients had depression, none of them received treatment indicating the lack of recognition of depressive symptoms. The need for early recognition and management of depression is to prevent further decline in quality of life, suicide attempts and progression of psoriasis. In our study there were 4 patients with suicidal ideations, literature shows 5 to 10 % of patients with psoriasis have suicidal ideations. This compels the need to address depression in these patients. Most of the patients in our study attributed the triggering factor of illness to psychological distress. Psychological distress also causes exacerbation of skin lesions and prolong the duration of psoriasis, but our study have not assessed in detail the psychological factors.

Patients with severe depression in our study mentioned that they had severe pruritis, feeling of being stigmatized in the society. Certain participants also believed that psoriasis was communicable and they were treated with caution

that their disease might spread to others with contact. Touch deprivation was significantly associated with impaired social and interpersonal relationships.

PREVALENCE AND SEVERITY OF ANXIETY

Prevalence of anxiety in psoriasis patients in our study is 20%, among them 16% had moderate anxiety requiring psychiatric intervention.

A study done by Fleming et al showed the prevalence of anxiety in psoriasis patients was 7 – 48%. Another study by Emily Mc Donough showed prevalence of anxiety was 36.6%. Here in our study we have got a prevalence that is lesser compared to other studies. Higher anxiety scores were present in majority of inpatients with psoriasis and in those with scalp and guttate psoriasis.

The reason for anxiety could be explained by patients apprehension about progression of disease, social perception by others, duration and outcome of treatment. Studies have also highlighted the presence of both depression and anxiety disorders in psoriasis patients. Our study showed that 20 % had both depression and anxiety. This shows patients with psoriasis diagnosed as depressive disorder are likely to have anxiety symptoms as well.

SOCIODEMOGRAPHIC PROFILE INFLUENCE ON PSYCHIATRIC DISORDER

Majority of the study population comprised of participants in 3rd and 5th decade of life and most of them were females. Majority of the Indian studies have

reported a male predominance of psoriasis, but in our study sample majority are females.

Sociodemographic variable have not been consistent in predicting the psychiatric morbidity. In our study also, there was only weak association of sociodemographic variables with depression and anxiety.

INFLUENCE OF SEVERITY OF PSORIASIS ON DEPRESSION AND ANXIETY

Patients with severe psoriasis had higher frequency of psychiatric morbidity. our study also revealed that patients with higher PASI score had severe depression and anxiety scores.

Majority of studies have reported positive correlation of severity of psoriasis with depression and anxiety. Our findings are also in agreement with previous studies. However our study results are in contrast to the study done by fortune et al (134), who reported that depression and anxiety was not influenced by psoriasis severity (135, 136)

Our study also found that there was positive correlation between severity of psoriasis and poor quality of life. The possible reasons could be visible skin lesions that impair the individual's self esteem and thereby interpersonal communication, systemic involvement causing occupational dysfunction, absenteeism.

Some studies have assessed the relationship between duration of psoriasis with depression and anxiety. Since our study sample contains study population with psoriasis for more than 5 years, significant association could not be ascertained in our study.

QUALITY OF LIFE IN PATIENTS WITH PSORIASIS In our study, out of 100 patients, quality of life was moderately to extremely affected in 84% of patients. There was positive correlation between HAM-D and HAM-A scores and DQLI. This shows that depressive and anxiety symptoms in psoriasis patients do play a major role in individual's functioning. Most of the patients in our study had reported that presence of skin lesions had restricted their previous pattern of clothing, for example – they had to wear only cotton dresses and full sleeves in order to cover their lesions. participants also reported that their social and leisure activities were affected, some of them had to restrict themselves from attending social gathering in view of being commented by others.

COMPARISION OF QUALITY OF LIFE AND SEVERITY OF PSORIASIS

Numerous studies have assessed the impact of psoriasis severity on the quality of life and the influence of depression and anxiety on severity of psoriasis. But only very few studies have examined the influence of disease severity on quality of life and psychiatric morbidity. This is one of the methodological

advantages that we have in our study. the proportion of patients with moderately to extremely large effect on quality of life was higher in patients with co morbid depression / anxiety. Patients with depression and anxiety had higher PASI score.

CONCLUSION

The results of this study is of particular interest, because it has highlighted the necessity of need for psychiatric evaluation of psoriasis patients. Psoriasis per say results in impairment in social and occupational domains. It also significantly causes interpersonal conflicts and lack of self esteem that needs to be addressed. Severe depression in these patients go unnoticed and could even result in suicide attempts. Our study also emphasises the fact that certain participants and their families considered psoriasis as a communicable disease leading to higher load of distress and stigma in the society. Patients, including their families have to be educated regarding the illness, and the need for being cared. Coping strategies to mitigate psychological stress have to be taught to these patients, since stress could exacerbate their skin lesions and also cause psychiatric illness. Timely intervention of psychiatric morbidity will improve the patient's condition and thereby the quality of life.

LIMITATIONS

- Study population from the same setting, small sample size. Large sample size and control group needed for generalisation of results. This is a cross sectional study, so impact of treatment on psychiatric morbidity whether it improves the quality of life and decrease the severity of psoriasis is not known.
- Arguing for the causality of depression and anxiety in psoriasis is also limited. Other common psychiatric morbidities like somatoform disorder, sleep disorder, substance dependence and sexual disorders are not assessed.
- Side effects of treatments used for psoriasis could also be associated with psychiatric morbidity.
- Personality traits, maladaptive schemas could also influence a persons vulnerability to develop mentall illness, which is not included in this study.

FUTURE DIRECTIONS

- Various types of psoriasis and its association with psychiatric morbidity need to be studied.
- Studies to explore the biochemical aspects of psoriasis and the role of inflammatory mediators on depression and anxiety before and after treatment with antidepressants.
- Studies to look for good outcome measures in psoriasis after treatment with SSRI, considering that SSRI downregulates T cell proliferation.

BIBLIOGRAPHY

1. Sullivan RL, Lipper G, Lerner EA. The neuro-immuno-cutaneous-endocrine network: Relationship of mind and skin. *Arch Dermatol* 1998;134:1431-5.
2. Katon WJ. Epidemiology and treatment of depression in patients with chronic medical illness. *Dialogues Clin Neurosci* 2011;13:7-23.
3. Johnson TJ, Basu S, Pisani BA, Avery EF, Mendez JC, Calvin JE Jr, *et al.* Depression predicts repeated heart failure hospitalizations. *J Card Fail* 2012;18:246-52.
4. Picardi A, Abeni D, Melchi CF, Puddu P, Pasquini P. Psychiatric morbidity in dermatological outpatients: An issue to be recognized. *Br J Dermatol* 2000;143:983-91.
5. S. K. Kurd, A. B. Troxel, P. Crits-Christoph, and J.M. Gelfand, "The risk of depression, anxiety, and suicidality in patients with psoriasis: a population-based cohort study, " *Archives of Dermatology*, vol. 146, no. 8, pp. 891–895, 2010.
6. M. Ghajarzadeh, M. Ghiasi, and S. Kheirkhah, "Associations between skin diseases and quality of life: a comparison of psoriasis, vitiligo, and alopecia areata, " *Acta Medica Iranica*, vol. 50, no. 7, pp. 511–515, 2012.

7. M. Karelson, H. Silm, and K. Kingo, "Quality of life and emotional state in vitiligo in an Estonian sample: comparison with psoriasis and healthy controls, " *Acta Dermato Venereologica*, vol. 93, no. 4, pp. 446–450, 2013.
8. V. Kumar, S. K. Mattoo, and S. Handa, "Psychiatric morbidity in pemphigus and psoriasis: a comparative study from India, " *Asian Journal of Psychiatry*, vol. 6, no. 2, pp. 151–156, 2013.
9. N. Sharma, R. V. Koranne, and R. K. Singh, "Psychiatric morbidity in psoriasis and vitiligo: a comparative study, " *Journal of Dermatology*, vol. 28, no. 8, pp. 419–423, 2001.
10. A. Mufaddel and A. E. Abdelgani, "Psychiatric comorbidity in patients with psoriasis, vitiligo, acne, eczema and group of patients with miscellaneous dermatological diagnoses, " *Open Journal of Psychiatry*, vol. 4, no. 3, pp. 168–175, 2014.
11. Kasenõnm P, Mesila I, Piirsoo A, Kull M, Mikelsaar M, Mikelsaar RH. Macroscopic oropharyngeal signs indicating impaired defensive function of palatine tonsils in adults suffering from recurrent tonsillitis. *APMIS* 2004;112: 248–256. doi: 10.1111/j. 1600-0463.2004.apm11204-0504.x
12. Skibinski G, Skibinska A, James K. Tonsil stromal cell lines expressing follicular dendritic cell-like properties--isolation, characterization and interaction with B lymphocytes. *Biochem Soc Trans* 1997;25:233S.

13. Steiniger B, Trabandt M, Barth PJ. The follicular dendritic cell network in secondary follicles of human palatine tonsils and spleens. *Histochemistry and Cell Biology* 2011;135:327-336. doi : 10.1007/s00418-011-0799-x.
14. Koch RJ, Brodsky L. Qualitative and quantitative immunoglobulin production by specific bacteria in chronic tonsillar disease. *Laryngoscope* 1995;105:42-48.
15. Brandtzaeg P, Jahnsen FL, Farstad IN. Immune functions and immunopathology of the mucosa of the upper respiratory pathways.? 1996;116 (2):149-159.
16. Bernstein JM, Ballow M, Rich G. Detection of intracytoplasmic cytokines by flow cytometry in adenoids and peripheral blood lymphocytes of children. *Ann Otol Rhinol Laryngol* 2001;110:442-446.
17. Farstad IN, Halstensen TS, Kvale D, Fausa O, Brandtzaeg P. Topographic distribution of homing receptors on B and T cells in human gut-associated lymphoid tissue: relation of L-selectin and integrin alpha 4 beta 7 to naive and memory phenotypes. *Am J Pathol* 1997;150:187-199.
18. Kilian M, Reinholdt J, Lomholt H, Poulsen K, Frandsen EV. Biological significance of IgA1 proteases in bacterial colonization and pathogenesis: critical evaluation of experimental evidence. *APMIS* 1996;104:321-338.

19. Fredriksson T, Pettersson U. Severe psoriasis – oral therapy with a new retinoid. *Dermatologica* 1978;157:238–244.
20. Mattozzi C, AG Richetta, Cantisani C, L Macaluso, Calvieri S. Psoriasis: New insight about pathogenesis, role of barrier organ integrity, NLR/CATERPILLER family genes and microbial flora. *Dermatol* 2012;39:752-60..
21. Honig PJ. Guttate psoriasis associated with perianal streptococcal disease. *J Pediatr* 1998;113: 1037 – 1039
22. Winfield JM. Psoriasis as a sequel to acute inflammations of the tonsils: a clinical note, *J Cutan Dis*, 1916;34: 441–443.
23. Trueb RM. Therapies for childhood psoriasis, *Curr Probl Dermatol*, 2009;38:137-159. Epub 2009 Jul 28.
24. Lomholt G. Prevalence of skin disease in a population: A census study from the Faroe islands. *Dan Med Bull* 1964;11:1–7.
25. Green AC. Australian Aborigines and psoriasis. *Australas J Dermatol* 1984;25:18–24.
26. Convit J. Investigation of the incidence of psoriasis amongst Latin-American Indians. In: *Proceedings of 13th Congress on Dermatology*. Amsterdam: Excerpta Medica, 1962:196.
27. Okhandiar RP, Banerjee BN. Psoriasis in the tropics: An epidemiological survey. *J Indian Med Assoc* 1963;41:550-6.

28. Bedi TR. Clinical profile of psoriasis in North India. *Indian J Dermatol Venereol Leprol* 1995;61:202-5.
29. Kaur I, Handa S, Kumar B. Natural history of psoriasis: a study from the Indian subcontinent. *J Dermatol* 1997;24:230-4.
30. Farber EM, Nall ML. The natural history of psoriasis in 5, 600 patients. *Dermatologica* 1974;148:1-18.
31. Bowcock AM, Krueger JG. Getting under the skin: the immunogenetics of psoriasis. *Nat Rev Immunol* 2005;5: 699-711. [Erratum, *Nat Rev Immunol* 2005;5:826.]
32. Trembath RC, Clough RL, Rosbotham JL, et al. Identification of a major susceptibility locus on chromosome 6p and evidence for further disease loci revealed by a two stage genome-wide search in psoriasis. *Hum Mol Genet* 1997;6:813-20.
33. Asumalahti K, Laitinen T, Itkonen- Vatjus R, et al. A candidate gene for psoriasis near HLA-C, HCR (Pg8), is highly polymorphic with a disease-associated susceptibility allele. *Hum Mol Genet* 2000;9:1533-42. [Erratum, *Hum Mol Genet* 2001;10:301.]
34. Allen MH, Veal C, Faassen A, et al. A non-HLA gene within the MHC in psoriasis. *Lancet* 1999;353:1589-90.

35. Wolf N, Quaranta M, Prescott NJ, et al. Psoriasis is associated with pleiotropic susceptibility loci identified in type II diabetes and Crohn disease. *J Med Genet* 2008;45:114-6.
36. Nair RP, Stuart PE, Nistor I, et al. Sequence and haplotype analysis supports HLA-C as the psoriasis susceptibility 1 gene. *Am J Hum Genet* 2006;78:827-51.
37. Asumalahti K, Ameen M, Suomela S, et al. Genetic analysis of PSORS1 distinguishes guttate psoriasis and palmoplantar pustulosis. *J Invest Dermatol* 2003;120: 627-32.
38. Allen MH, Ameen H, Veal C, et al. The major psoriasis susceptibility locus PSORS1 is not a risk factor for late-onset psoriasis. *J Invest Dermatol* 2005;124: 103-6.
39. Chablani UA, Contractor NM, Gadgil RB. HLA and complement C4 studies in psoriasis vulgaris. *Natl Med J India* 1992;5:8-11.
40. Pitchappan RM, Koteeswaran A, Kakkaniah VN, Manickasundari M, Rajaram V, Muthuveeralakshmi P, *et al.* HLA Bw57 and DR7 association with psoriasis vulgaris in south India. *Tissue Antigens* 1989;34:133-7.
41. Rani R, Narayan R, Fernandez-Vina MA, Stastny P. Role of HLA-B and C alleles in development of psoriasis in patients from North India. *Tissue Antigens* 1998;51:618-22.

42. Farber EM, Jacobs AH. Infantile psoriasis. *Am J Dis Child* 1977;131:1266-9.
43. Farber EM, Carlsen RA. Psoriasis in childhood. *Calif Med* 1966;105:415-20.
44. Hansen AG. Psoriasis in childhood. In: Farber EM, Cox AJ, editors. *Psoriasis: Proceedings of the International Symposium*. Stanford, CA: Stanford University Press, 1971. p. 53–9.
45. Bedi TR. Clinical profile of psoriasis in North India. *Indian J Dermatol Venereol Leprol* 1995;61:202-5
46. Kaur I, Handa S, Kumar B. Natural history of psoriasis: a study from the Indian subcontinent. *J Dermatol* 1997;24:230-4.
47. Kimball AB, Gieler U, Linder D, Sampogna F, Warren RB, Augustin M. Psoriasis: is the impairment to a patient's life cumulative? *J Eur Acad Derm Venereol* 2010; 24: 989–1004.
48. Warren RB, Kleyn CE, Gulliver WP. Cumulative life course impairment in psoriasis: patient perception of disease-related impairment throughout the life course. *Br J Dermatol* 2011; 164 (Suppl 1): 1–14.
49. Frank JD. *Persuasion and Healing*. Johns Hopkins University Press: Baltimore. 1961.

50. Friedman M, Rosenman RH. Association of specific overt behavior pattern with blood and cardiovascular findings; blood cholesterol level, blood clotting time, incidence of atherosclerosis, and clinical coronary artery disease. *JAMA* 1959; 169: 1286–1296.
51. Picardi A, Pasquini P, Abeni D, Fassone G, Mazzotti E, Fava GA. Psychosomatic assessment of skin diseases in clinical practice. *Psychother Psychosom* 2005; 74: 315–322.
52. A. Garg, M.-M. Chren, L. P. Sands et al., “Psychological stress perturbs epidermal permeability barrier homeostasis: implications for the pathogenesis of stress-associated skin disorders, ” *Archives of Dermatology*, vol. 137, no. 1, pp. 53–59, 2001.
53. Naylor C, Parsonage M, McDaid D, Knapp M, Fossey M, Long term conditions and mental health: King’s fund and centre for mental health. 2012
54. S. R. Rapp, S. R. Feldman, M. L. Exum, A. B. Fleischer, and D. M. Reboussin, “Psoriasis causes as much disability as other major medical diseases, ” *Journal of the American Academy of Dermatology*, vol. 41, no. 3, part 1, pp. 401–407, 1999.
55. Y. Dowlati, N. Herrmann, W. Swardfager et al., “A meta-analysis of cytokines in major depression, ” *Biological Psychiatry*, vol. 67, no. 5, pp. 446–457, 2010.

56. C. Pizzi, S. Mancini, L. Angeloni, F. Fontana, L. Manzoli, and G. M. Costa, "Effects of selective serotonin reuptake inhibitor therapy on endothelial function and inflammatory markers in patients with coronary heart disease," *Clinical Pharmacology and Therapeutics*, vol. 86, no. 5, pp. 527–532, 2009.
57. A. Steptoe, M. Hamer, and Y. Chida, "The effects of acute psychological stress on circulating inflammatory factors in humans: a review and meta-analysis," *Brain, Behavior, and Immunity*, vol. 21, no. 7, pp. 901–912, 2007.
58. M. A. Gupta, A. K. Gupta, and H. F. Haberman, "Psoriasis and psychiatry: an update," *General Hospital Psychiatry*, vol. 9, no. 3, pp. 157–166, 1987.
59. M. Joëls and E. R. de Kloet, "Mineralocorticoid and glucocorticoid receptors in the brain. Implications for ion permeability and transmitter systems," *Progress in Neurobiology*, vol. 43, no. 1, pp. 1–36, 1994.
60. L. Arborelius, M. J. Owens, P. M. Plotsky, and C. B. Nemeroff, "The role of corticotropin-releasing factor in depression and anxiety disorders," *Journal of Endocrinology*, vol. 160, no. 1, pp. 1–12.
61. B. Zbytek, A. Mysliwski, A. Slominski, J. Wortsman, E. T. Wei, and J. Mysliwska, "Corticotropin-releasing hormone affects cytokine production in human HaCaT keratinocytes," *Life Sciences*, vol. 70, no. 9, pp. 1013–1021, 2002.

62. M.-E. Quevedo, A. Slominski, W. Pinto, E. Wel, and J. Wortsman, "Pleiotropic effects of corticotropin releasing hormone on normal human skin keratinocytes, " *In Vitro Cellular and Developmental Biology: Animal*, vol. 37, no. 1, pp. 50–54, 2001.
63. B. Zbytek, L. M. Pfeffer, and A. T. Slominski, "Corticotropin-releasing hormone stimulates NF- κ B in human epidermal keratinocytes, " *Journal of Endocrinology*, vol. 181, no. 3, pp. R1–R7, 2004.
64. J. E. Kim, D. H. Cho, H. S. Kim et al., "Expression of the corticotropin-releasing hormone-proopiomelanocortin axis in the various clinical types of psoriasis, " *Experimental Dermatology*, vol. 16, no. 2, pp. 104–109, 2007., 1999.
65. H. L. Richards, D. W. Ray, B. Kirby et al., "Response of the hypothalamic-pituitary-adrenal axis to psychological stress in patients with psoriasis, " *British Journal of Dermatology*, vol. 153, no. 6, pp. 1114–1120, 2005.
66. A. Garg, M.-M. Chren, L. P. Sands et al., "Psychological stress perturbs epidermal permeability barrier homeostasis: implications for the pathogenesis of stress-associated skin disorders, " *Archives of Dermatology*, vol. 137, no. 1, pp. 53–59, 2001.
67. S. D. Kreibitz, "Autonomic nervous system activity in emotion: a review, " *Biological Psychology*, vol. 84, no. 3, pp. 394–421, 2010.

68. A. Z. Scalco, M. U. P. B. Rondon, I. C. Trombetta et al., “Muscle sympathetic nervous activity in depressed patients before and after treatment with sertraline, ” *Journal of Hypertension*, vol. 27, no. 12, pp. 2429–2436, 2009.
69. D. A. Barton, T. Dawood, E. A. Lambert et al., “Sympathetic activity in major depressive disorder: identifying those at increased cardiac risk?” *Journal of Hypertension*, vol. 25, no. 10, pp. 2117–2124, 2007.
70. R. C. Veith, N. Lewis, O. A. Linares et al., “Sympathetic nervous system activity in major depression: basal and desipramine-induced alterations in plasma norepinephrine kinetics, ” *Archives of General Psychiatry*, vol. 51, no. 5, pp. 411–422, 1994.
71. M. V. Singh, M.W. Chapleau, S. C. Harwani, and F. M. Abboud, “The immune system and hypertension, ” *Immunologic Research*, vol. 59, no. 1–3, pp. 243–253, 2014.
72. K. M. Grebe, K. Takeda, H. D. Hickman et al., “Cutting edge: sympathetic nervous system increases proinflammatory cytokines and exacerbates influenza A virus pathogenesis, *Journal of Immunology*, vol. 184, no. 2, pp. 540–544, 2010.
73. Y. Katayama, M. Battista, W.-M. Kao et al., “Signals from the sympathetic nervous system regulate hematopoietic stem cell egress from bone marrow, ” *Cell*, vol. 124, no. 2, pp. 407–421, 2006.

74. P. Saint-Mezard, C. Chavagnac, S. Bosset et al., "Psychological stress exerts an adjuvant effect on skin dendritic cell functions in vivo, " *The Journal of Immunology*, vol. 171, no. 8, pp. 4073–4080, 2003
75. C. L. Lubahn, D. Lorton, J. A. Schaller, S. J. Sweeney, and D.L. Bellinger, "Targeting α - and β -adrenergic receptors differentially shifts Th1, Th2, and inflammatory cytokine profiles in immune organs to attenuate adjuvant arthritis, " *Frontiers in Immunology*, vol. 5, article 346, 2014.
76. K. H. Basavaraj, N. M. Ashok, R. Rashmi, and T. K. Praveen, "The role of drugs in the induction and/or exacerbation of psoriasis, " *International Journal of Dermatology*, vol. 49, no. 12, pp. 1351–1361, 2010.
77. S. Wu, J. Han, W.-Q. Li, and A. A. Qureshi, "Hypertension, antihypertensive medication use, and risk of psoriasis, " *JAMA Dermatology*, vol. 150, no. 9, pp. 957–963, 2014.
78. D. Kovacs, M. Falchi, G. Cardinali et al., "Immunohistochemical analysis of keratinocyte growth factor and fibroblast growth factor 10 expression in psoriasis, " *Experimental Dermatology*, vol. 14, no. 2, pp. 130–137, 2005.

79. N. Yao, J.-X. Xia, X.-M. Liu et al., “Topical application of a new monoclonal antibody against fibroblast growth factor 10 (FGF 10)mitigates propranolol-induced psoriasis-like lesions in guinea pigs, ” *European Review forMedical and Pharmacological Sciences*, vol. 18, no. 7, pp. 1085–1091, 2014.
80. F. S. Dhabhar, “Effects of stress on immune function: the good, the bad, and the beautiful, ” *Immunologic Research*, vol. 58, no. 2-3, pp. 193–210, 2014.
81. F. S. Dhabhar, “Enhancing versus suppressive effects of stress on immune function: implications for immunoprotection and immunopathology, ” *NeuroImmunoModulation*, vol. 16, no. 5, pp. 300–317, 2009.
82. J. E. Carroll, C. A. Low, A. A. Prather et al., “Negative affective responses to a speech task predict changes in interleukin (IL)-6, ” *Brain, Behavior, and Immunity*, vol. 25, no. 2, pp. 232–238, 2011.
83. E. Puterman, E. S. Epel, A. O’Donovan, A. A. Prather, K Aschbacher, and F. S. Dhabhar, “Anger is associated with increased IL-6 stress reactivity in women, but only among those low in social support, ” *International Journal of Behavioral Medicine*, vol. 21, no. 6, pp. 936–945, 2013.
84. M. J. Maher, P. A. Mora, and H. Leventhal, “Depression as a predictor of perceived social support and demand: a componential approach using a

- prospective sample of older adults, ” *Emotion*, vol. 6, no. 3, pp. 450–458, 2006.
85. E. Stice, P. Rohde, J. Gau, and C. Ochner, “Relation of depression to perceived social support: results from a randomized adolescent depression prevention trial, ” *Behaviour Research and Therapy*, vol. 49, no. 5, pp. 361–366, 2011.
 86. S. Sephton and D. Spiegel, “Circadian disruption in cancer: a neuroendocrine-immune pathway from stress to disease?” *Brain, Behavior, and Immunity*, vol. 17, no. 5, pp. 321–328, 2003.
 87. AKAY A, PEKCANLAR A, BOZDAG KE, ALTINTAS L, KARAMANA, *J Eur Acad Dermatol Venereol*, 16 (2002) 347.
 88. KIRBY B, RICHARDS HL, WOO P, HINDLE E, MAIN CJ, GRIFFITHS CE, *J Am Acad Dermatol*, 45 (2001) 72
 89. Sharma N, Koranne RV, Singh RK. Psychiatric morbidity in psoriasis and vitiligo: a comparative study. *J Dermatol*. 2001;28 (8):419-23.
 90. Mattoo SK, Handa S, Kaur I, Gupta N, Malhotra R. Psychiatric morbidity in vitiligo and psoriasis: a comparative study from India. *J Dermatol*. 2001;28 (8):424-32
 91. Braathen LR, Botten G, Bjerkedal T. Psoriatics in Norway. A questionnaire study on health status, contact with paramedical

- professions, and alcohol and tobacco consumption. *Acta Dermato-Venereologica* (Suppl) 1989; 142:9-12.
92. Lindegard B. Disease associated with psoriasis in a general population of 159, 200 middle-aged urban, native Swedes. *Dermatologica* 1986;172:298-304. |
 93. Mills CM, Srivastava ED. Harvey IM Smoking habits in psoriasis: a case controlled study. *Br J Dermatology* 1992;127: 18-21. |
 94. A Cross-sectional Study of Prevalence and Implications of Depression and Anxiety in Indian J Psychol Med. 2015 Oct-Dec; 37 (4): 434–440.
 95. Sreelatha Lakshmy, Sivaprakash Balasundaram, Sukanto Sarkar, A cross sectional study of prevalence and implication of depression and anxiety in psoriasis : *Indian journal of psychological medicine* 2015; 37:434-40
 96. L. B. Kartha, L. Chandrashekar, M. Rajappa et al., “Serum melatonin levels in psoriasis and associated depressive symptoms, ” *Clinical Chemistry and Laboratory Medicine*, vol. 52, no. 6, pp. e123–e125, 2014.
 97. A. Carrillo-Vico, P. J. Lardone, N. ´ Alvarez-´Snchez, A. Rodr´ıguez-Rodr´ıguez, and J. M. Guerrero, “Melatonin: buffering the immune system, ” *International Journal of Molecular Sciences*, vol. 14, no. 4, pp. 8638–8683, 2013

98. E. Esposito and S. Cuzzocrea, "Antiinflammatory activity of melatonin in central nervous system, " *Current Neuropharmacology*, vol. 8, no. 3, pp. 228–242, 2010.
99. A. Miles and D. Philbrick, "Melatonin: perspectives in laboratory medicine and clinical research, " *Critical Reviews in Clinical Laboratory Sciences*, vol. 25, no. 3, pp. 231–253, 1987.
100. N. Mozzanica, G. Tadini, A. Radaelli et al., "Plasma melatonin levels in psoriasis, " *Acta Dermato-Venereologica*, vol. 68, no. 4, pp. 312–316, 1988.
101. K. R. Feingold and P. M. Elias, "Endocrine-skin interactions. Cutaneous manifestations of adrenal disease, pheochromocytomas, carcinoid syndrome, sex hormone excess and deficiency, polyglandular autoimmune syndromes, multiple endocrine neoplasia syndromes, and other miscellaneous disorders, " *Journal of the American Academy of Dermatology*, vol. 19, no. 1, part1, pp. 1–20, 1988.
102. S. N. Goyal, D. M. Kokare, C. T. Chopde, and N. K. Subhedar, "Alpha-melanocyte stimulating hormone antagonizes antidepressant-like effect of neuropeptide Y in Porsolt's test in rats, " *Pharmacology Biochemistry and Behavior*, vol. 85, no. 2, pp. 369–377, 2006.
103. D. M. Kokare, M. P. Dandekar, P. S. Singru, G. L. Gupta, and N. K. Subhedar, "Involvement of alpha-MSH in the social isolation

- induced anxiety- and depression-like behaviors in rat, ”
Neuropharmacology, vol. 58, no. 7, pp. 1009–1018, 2010.
104. R. H. Ehrensing and A. J. Kastin, “Melanocyte-stimulating hormone-release inhibiting hormone as an antidepressant. A pilot study, ”
Archives of General Psychiatry, vol. 30, no. 1, pp. 63–65, 1974.
 105. M.-S. Lee, R.-Y. Lin, and M.-S. Lai, “Increased risk of diabetes mellitus in relation to the severity of psoriasis, concomitant medication, and comorbidity: a nationwide population-based cohort study, ” *Journal of the American Academy of Dermatology*, vol. 70, no. 4, pp. 691–698, 2014.
 106. E. Peschke and E. Mühlbauer, “New evidence for a role of melatonin in glucose regulation, ” *Best Practice & Research Clinical Endocrinology&Metabolism*, vol. 24, no. 5, pp. 829–841, 2010.
 107. R. Johnson, L. Staiano-Coico, L. Austin, I. Cardinale, R. Nabeya-Tsukifuji, and J. G. Krueger, “PUVA treatment selectively induces a cell cycle block and subsequent apoptosis in human T-lymphocytes, ”
Photochemistry and Photobiology, vol. 63, no. 5, pp. 566–571, 1996.
 108. T. J. Laing, B. C. Richardson, M. B. Toth, E. M. Smith, and R. M. Marks, “Ultraviolet light and 8-methoxypsoralen inhibit expression of endothelial adhesion molecules, ” *The Journal of Rheumatology*, vol. 22, no. 11, pp. 2126–2131, 1995.

109. G. Sethi and A. Sodhi, "Role of p38 mitogen-activated protein kinase and caspases in UV-B-induced apoptosis of murine peritoneal macrophages," *Photochemistry and Photobiology*, vol.79, no. 1, pp. 48–54, 2004.
110. Gupta MA, Gupta AK, Kirkby S, Schork NJ, Gorr SK, Ellis CN, et al. A psychocutaneous profile of psoriasis patients who are stress reactors. A study of 127 patients. *Gen Hosp Psychiatry*. 1989;11:166–73.
111. Sreelatha Lakshmy, Sivaprakash Balasundaram, Sukanto Sarkar, A cross sectional study of prevalence and implication of depression and anxiety in psoriasis : *Indian journal of psychological medicine* 2015; 37:434-40
112. Buske-Kirschbaum A, Ebrecht M, Kern S, Hellhammer DH. Endocrine stress responses in TH1-mediated chronic inflammatory skin disease (psoriasis vulgaris)--do they parallel stress-induced endocrine changes in TH2-mediated inflammatory dermatoses.*Psychoneuroendocrinology*. 2006;31:439–46.
113. Evers AWM, Verhoeven EWM, Kraaijmaat FW, de Jong EMGJ, de Brouwer SJM, Schalkwijk J, et al. How stress gets under the skin: cortisol and stress reactivity in psoriasis. *Br J Dermatol*. 2010;163:986–91.

114. Fortune DG, Richards HL, Griffiths CEM. Psychologic factors in psoriasis: consequences, mechanisms, and interventions. *Dermatol Clin.* 2005;23:681–94.
115. Nasreen S, Ahmed I, Effendi S. Frequency and magnitude of anxiety and depression in patients with psoriasis vulgaris. *J Coll Physicians Surg Pak.* 2008;18:397–400.
116. Hedlund JL, Viewig BW (1979) The Hamilton rating scale for depression: a comprehensive review. *Journal of Operational Psychiatry* **10**:149–165
117. Hamilton, M (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery, and Psychiatry.* **23**: 56–62
118. Hamilton M (1966) Assessment of change in psychiatric state by means of rating scales. *Proceedings of the Royal Society of Medicine* **59** (Suppl. 1): 10–13
119. Hamilton, M (1967) Development of a rating scale for primary depressive illness. *British Journal of Social and Clinical Psychology* **6**: 278–96
120. Hamilton, M (1969) Standardised assessment and recording of depressive symptoms. *Psychiatra, Neurologia, Neurochirurgia.* **72**:201–205

121. Hamilton, M (1980) Rating depressive patients. *Journal of Clinical Psychiatry*. **41**: 21–24
122. Maier W, Buller R, Philipp M, Heuser I. The Hamilton Anxiety Scale: reliability, validity and sensitivity to change in anxiety and depressive disorders. *J Affect Disord* 1988;14 (1):61–8.
123. McDowell, I., Newell, C., & McDowell, I. (2006). Measuring health: a guide to rating scales and questionnaires (Vol. 268). New York: Oxford University Press.
124. Finlay AY, Khan GK. "The Dermatology Life Quality Index: A simple practical measure for routine clinical use". British Association of Dermatologists Annual Meeting, Oxford, July 1993. *British Journal of Dermatology*, 1993; 129 (Suppl 42): 27.
125. Finlay AY, Khan GK. "Dermatology Life Quality Index (DLQI) - a simple practical measure for routine clinical use". *Clinical and Experimental Dermatology*, 1994; 19: 210-216.
126. Khilji FA, Gonzalez M, Finlay AY. Clinical meaning of change in Dermatology Life Quality Index scores. *British Journal of Dermatology* 2002; 147 (Suppl 62): 50
127. Verall JE, Gorham DR (1962). The brief psychiatric rating scale. *Psychological Reports* 1962 vol. 10, pp799-812

128. Akay A, Pekcanlar A, Bozdog KE, Altintas L, Karaman A. Assessment of depression in subjects with psoriasis vulgaris and lichen planus. *J Eur Acad Dermatol Venereol*. 2002;16:347–52.
129. Devrimci-Ozguven H, Kundakci TN, Kumbasar H, Boyvat A. The depression, anxiety, life satisfaction and affective expression levels in psoriasis patients. *J Eur Acad Dermatol Venereol*. 2000;14:267–71.
130. Dowlatshahi EA, Wakkee M, Arends LR, Nijsten T. The prevalence and odds of depressive symptoms and clinical depression in psoriasis patients: a systematic review and meta-analysis. *J Invest Dermatol*. 2014;134:1542–51.
131. Nasreen S, Ahmed I, Effendi S. Frequency and magnitude of anxiety and depression in patients with psoriasis vulgaris. *J Coll Physicians Surg Pak*. 2008;18:397–400.
132. Rabin F, Bhuiyan SI, Islam T, Haque MA, Islam MA. Psychiatric and psychological comorbidities in patients with psoriasis- a review. *Mymensingh Med J*. 2012;21:780–6.
133. Sampogna F, Chren MM, Melchi CF, Pasquini P, Tabolli S, Abeni D. Age gender, quality of life and psychological distress in patients hospitalized with psoriasis. *Br J Dermatol*. 2006;154:325–31.
134. Fortune DG, Richards HL, Griffiths CEM, Main CJ. Psychological stress, distress and disability in patients with psoriasis:

consensus and variation in the contribution of illness perceptions, coping and alexithymia. *Br J Clin Psychol.* 2002;41 (Pt 2):157–74.

135. Rieder E, Tausk F. *Psoriasis, a model of dermatologic psychosomatic disease: psychiatric implications and treatments. Int J Dermatol.* 2012;5:12–26.
136. Magin PJ, Pond CD, Smith WT, Watson AB, Goode SM. *Correlation and agreement of self-assessed and objective skin disease severity in a cross-sectional study of patients with acne, psoriasis, and atopic eczema. Int J Dermatol.* 2011;50:1486–90.

APPENDIX

Information to Participants

Title: Depression, Anxiety and Quality of life in patients with psoriasis : a cross sectional study

Principal Investigator: Dr. ROSE MONICA V. R

Name of Participant:

Site: Department of Dermatology, Madras Medical College, Chennai

You are invited to take part in this research. The information in this document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns.

What is the purpose of research?

The prevalence of depression and anxiety in those with psoriasis is higher and the quality of life is low . if these comorbidities are not treated , they influence the treatment improvement of primary dermatological condition .Timely intervention will improve the patient's dermatological disease and thereby the quality of life

The study design

Study will be done at Department of Dermatology, Madras medical college. 100 consecutive patients who are physically stable, giving their consent will be taken up for the study. Quality of life , Prevalence and severity of depression and Anxiety will be assessed in patients with psoriasis .

Study Procedures

1. Study will include a population of patients diagnosed with psoriasis attending outpatient and inpatient of Department of Dermatology. They are chosen for study if they are physically stable after taking an informed consent. Later, standard assessment tools, i.e., Hamilton rating scale for depression (HAM-D), Hamilton rating scale for anxiety (HAM-A), Dermatology life quality index (DLQI), Brief psychiatric rating scale (BPRS) scale are used to assess the Quality of life, Prevalence and severity of Depression and Anxiety. Various data obtained will be analyzed statistically to get the results.

. Confidentiality of the information obtained from you

You have the right to confidentiality regarding the privacy of your medical information (personal details, results of physical examinations, investigations, and your medical history). By signing this document, you will be allowing the research team investigators, other study personnel, Institutional Ethics Committee and any person or agency required by law like the Drug Controller General of India to view your data, if required.

The information from this study, if published in scientific journals or presented at scientific meetings, will not reveal your identity.

How will your decision to not participate in the study affect you?

Your decision not to participate in this research study will not affect your medical care or your relationship with the investigator or the institution. You will be taken care of and you will not lose any benefits to which you are entitled.

Can you decide to stop participating in the study once you start?

The participation in this research is purely voluntary and you have the right to withdraw from this study at any time during the course of the study without giving any reasons. However, it is advisable that you talk to the research team prior to discontinuing from the study.

Signature of Investigator
of Participant

Signature

of the Guardian

Signature

Date

Date

INFORMED CONSENT FORM

(This is only a guideline – Relevant changes to be made as per the study requirements)

Title of the study:” Depression, Anxiety and Quality of life in patients with psoriasis : a cross sectional study

“ _____ ”.

Name of the Participant:

_____.

Name of the Principal (Co-Investigator): _Dr.ROSE MONICA V .R

_____.

Name of the Institution: Institute of mental health

_____.

Name and address of the sponsor / agency (ies) (if any):__No _____

_____.

Documentation of the informed consent

I _____ have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over 18 years of age and, exercising my free power of choice, hereby give my consent to be included as a participant in

’ Depression, Anxiety and Quality of life in patients with Psoriasis : a cross sectional study ’

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.
4. I have been explained about my rights and responsibilities by the investigator.
5. I have been informed the investigator of all the treatments I am taking or have taken in the past _____ months including any native (alternative) treatment.
6. I have been advised about the risks associated with my participation in this study.*

7. I have not participated in any research study within the past _____month(s). *
8. I have not donated blood within the past _____ months—Add if the study involves extensive blood sampling. *
9. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital. *
10. I am also aware that the investigator may terminate my participation in the study at any time, for any reason, without my consent. *
11. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.
12. I have understand that my identity will be kept confidential if my data are publicly presented
13. I have had my questions answered to my satisfaction.
14. I have decided to be in the research study.

I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

For adult participants:

Name and signature / thumb impression of the participant (or legal representative if participant incompetent)

Name _____ Signature _____
Date _____

Name and Signature of impartial witness (required for illiterate patients):

Name _____ Signature _____
Date _____

Address and contact number of the impartial witness:

Name and Signature of the investigator or his representative obtaining consent:

Name _____ Signature _____

Date _____

ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி தலைப்பு:

சோரியாசிஸ் நோயினால் வரும் மன அழுத்தம், மனப்பதற்றம் மற்றும் நோயாளியின் வாழ்க்கை தரத்தைப் பற்றிய குறுக்கு வெட்டு ஆய்வு

பங்கு கொள்பவர் பெயர் :

ஆராய்ச்சியாளர் : மரு.வா.ரோ.ரோஸ் மோனிகா

இடம் : ராஜீவ்காந்தி அரசு மருத்துவமனை.
சென்னை

நான் எனக்கு கொடுக்கப்பட்ட தகவல் தாளினை படித்து புரிந்துகொண்டேன்.

எனக்கு இந்த ஆராய்ச்சியின் ஒப்புதல் படிவம் விளக்கப்பட்டது.

எனக்கு இந்த ஆராய்ச்சியின் நோக்கமும், விவரங்களும் விளக்கப்பட்டது.

எனக்கு என்னுடைய உரிமைகளை பற்றி விளக்கப்பட்டது.

நான் இதற்கு முன்பு எடுத்துக்கொண்ட அனைத்து மருத்துவ முறைகளைப் பற்றி தெரிவித்திருக்கிறேன்.

இந்த ஆராய்ச்சியில் இருந்து நான் எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துகொண்டேன்.

என்னை பற்றிய எந்த தகவல்களும் அடையாளமும் வெளியிடப்படமாட்டாது என்பதை நான் புரிந்துகொண்டேன்

என்னுடைய முழு சுதந்திரத்துடன் இந்த ஆராய்ச்சியில் என்னைச் சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

பங்கேற்பாளர் பெயர் மற்றும் கையொப்பம் தேதி.....

பாதுகாவலர் பெயர் மற்றும் கையொப்பம் தேதி.....

ஆராய்ச்சியாளரின் பெயர் மற்றும் கையொப்பம் தேதி.....

HAMILTON DEPRESSION RATING SCALE (HAM-D)

(To be administered by a health care professional)

Patient Name _____

Today's Date _____

The HAM-D is designed to rate the severity of depression in patients. Although it contains 21 areas, calculate the patient's score on the first 17 answers.

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1. DEPRESSED MOOD

(Gloomy attitude, pessimism about the future, feeling of sadness, tendency to weep)

- 0 = Absent
 - 1 = Sadness, etc.
 - 2 = Occasional weeping
 - 3 = Frequent weeping
 - 4 = Extreme symptoms
-

☐

2. FEELINGS OF GUILT

- 0 = Absent
 - 1 = Self-reproach, feels he/she has let people down
 - 2 = Ideas of guilt
 - 3 = Present illness is a punishment; delusions of guilt
 - 4 = Hallucinations of guilt
-

☐

3. SUICIDE

- 0 = Absent
 - 1 = Feels life is not worth living
 - 2 = Wishes he/she were dead
 - 3 = Suicidal ideas or gestures
 - 4 = Attempts at suicide
-

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4. INSOMNIA - Initial

(Difficulty in falling asleep)

- 0 = Absent
 - 1 = Occasional
 - 2 = Frequent
-

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5. INSOMNIA - Middle

(Complains of being restless and disturbed during the night. Waking during the night.)

- 0 = Absent
 - 1 = Occasional
 - 2 = Frequent
-

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6. INSOMNIA - Delayed

(Waking in early hours of the morning and unable to fall asleep again)

- 0 = Absent
 - 1 = Occasional
 - 2 = Frequent
-

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7. WORK AND INTERESTS

- 0 = No difficulty
 - 1 = Feelings of incapacity, listlessness, indecision and vacillation
 - 2 = Loss of interest in hobbies, decreased social activities
 - 3 = Productivity decreased
 - 4 = Unable to work. Stopped working because of present illness only. (Absence from work after treatment or recovery may rate a lower score).
-

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8. RETARDATION

(Slowness of thought, speech, and activity; apathy; stupor.)

- 0 = Absent
 - 1 = Slight retardation at interview
 - 2 = Obvious retardation at interview
 - 3 = Interview difficult
 - 4 = Complete stupor
-

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9. AGITATION

(Restlessness associated with anxiety.)

- 0 = Absent
 - 1 = Occasional
 - 2 = Frequent
-

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10. ANXIETY - PSYCHIC

- 0 = No difficulty
- 1 = Tension and irritability
- 2 = Worrying about minor matters
- 3 = Apprehensive attitude
- 4 = Fears

HAMILTON DEPRESSION RATING SCALE (HAM-D)

(To be administered by a health care professional)

- ☐ **11. ANXIETY - SOMATIC**
Gastrointestinal, indigestion
Cardiovascular, palpitation, Headaches
Respiratory, Genito-urinary, etc.
0 = Absent
1 = Mild
2 = Moderate
3 = Severe
4 = Incapacitating

- ☐ **12. SOMATIC SYMPTOMS - GASTROINTESTINAL**
(Loss of appetite, heavy feeling in abdomen; constipation)
0 = Absent
1 = Mild
2 = Severe

- ☐ **13. SOMATIC SYMPTOMS - GENERAL**
(Heaviness in limbs, back or head; diffuse backache; loss of energy and fatigability)
0 = Absent
1 = Mild
2 = Severe

- ☐ **14. GENITAL SYMPTOMS**
(Loss of libido, menstrual disturbances)
0 = Absent
1 = Mild
2 = Severe

- ☐ **15. HYPOCHONDRIASIS**
0 = Not present
1 = Self-absorption (bodily)
2 = Preoccupation with health
3 = Querulous attitude
4 = Hypochondriacal delusions

- ☐ **16. WEIGHT LOSS**
0 = No weight loss
1 = Slight
2 = Obvious or severe

- ☐ **17. INSIGHT**
(Insight must be interpreted in terms of patient's understanding and background.)
0 = No loss
1 = Partial or doubtful loss
2 = Loss of insight

TOTAL ITEMS 1 TO 17: _____
0 - 7 = Normal
8 - 13 = Mild Depression
14 - 18 = Moderate Depression
19 - 22 = Severe Depression
≥ 23 = Very Severe Depression

- ☐ **18. DIURNAL VARIATION**
(Symptoms worse in morning or evening. Note which it is.)
0 = No variation
1 = Mild variation; AM () PM ()
2 = Severe variation; AM () PM ()

- ☐ **19. DEPERSONALIZATION AND DEREALIZATION**
(feelings of unreality, nihilistic ideas)
0 = Absent
1 = Mild
2 = Moderate
3 = Severe
4 = Incapacitating

- ☐ **20. PARANOID SYMPTOMS**
(Not with a depressive quality)
0 = None
1 = Suspicious
2 = Ideas of reference
3 = Delusions of reference and persecution
4 = Hallucinations, persecutory

- ☐ **21. OBSESSIONAL SYMPTOMS**
(Obsessive thoughts and compulsions against which the patient struggles)
0 = Absent
1 = Mild
2 = Severe

* Adapted from Hamilton, M. *Journal of Neurology, Neurosurgery, and Psychiatry*. 23:56-62, 1960.

Hamilton Anxiety Rating Scale (HAM-A)

Below is a list of phrases that describe certain feeling that people have. Rate the patients by finding the answer which best describes the extent to which he/she has these conditions. Select one of the five responses for each of the fourteen questions.

0 = Not present, 1 = Mild, 2 = Moderate, 3 = Severe, 4 = Very severe.

1 Anxious mood ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Worries, anticipation of the worst, fearful anticipation, irritability.

2 Tension ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Feelings of tension, fatigability, startle response, moved to tears easily, trembling, feelings of restlessness, inability to relax.

3 Fears ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Of dark, of strangers, of being left alone, of animals, of traffic, of crowds.

4 Insomnia ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Difficulty in falling asleep, broken sleep, unsatisfying sleep and fatigue on waking, dreams, nightmares, night terrors.

5 Intellectual ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Difficulty in concentration, poor memory.

6 Depressed mood ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Loss of interest, lack of pleasure in hobbies, depression, early waking, diurnal swing.

7 Somatic (muscular) ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Pains and aches, twitching, stiffness, myoclonic jerks, grinding of teeth, unsteady voice, increased muscular tone.

8 Somatic (sensory) ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Tinnitus, blurring of vision, hot and cold flushes, feelings of weakness, pricking sensation.

9 Cardiovascular symptoms ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Tachycardia, palpitations, pain in chest, throbbing of vessels, fainting feelings, missing beat.

10 Respiratory symptoms ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Pressure or constriction in chest, choking feelings, sighing, dyspnea.

11 Gastrointestinal symptoms ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Difficulty in swallowing, wind abdominal pain, burning sensations, abdominal fullness, nausea, vomiting, borborygmi, looseness of bowels, loss of weight, constipation.

12 Genitourinary symptoms ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Frequency of micturition, urgency of micturition, amenorrhea, menorrhagia, development of frigidity, premature ejaculation, loss of libido, impotence.

13 Autonomic symptoms ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Dry mouth, flushing, pallor, tendency to sweat, giddiness, tension headache, raising of hair.

14 Behavior at interview ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4

Fidgeting, restlessness or pacing, tremor of hands, furrowed brow, strained face, sighing or rapid respiration, facial pallor, swallowing, etc.

DERMATOLOGY LIFE QUALITY INDEX (DLQI)

Hospital No: . Date: .
 Name: . Score: .
 Address: . Diagnosis: .

The aim of this questionnaire is to measure how much your skin problem has affected your life
OVER THE LAST WEEK. Please tick (✓) one box for each question.

- | | | |
|---|-------------------------------------|---------------------------------------|
| 1. Over the last week, how itchy, sore, painful or stinging has your skin been? | Very much <input type="checkbox"/> | |
| | A lot <input type="checkbox"/> | |
| | A little <input type="checkbox"/> | |
| | Not at all <input type="checkbox"/> | |
| 2. Over the last week, how embarrassed or self conscious have you been because of your skin? | Very much <input type="checkbox"/> | |
| | A lot <input type="checkbox"/> | |
| | A little <input type="checkbox"/> | |
| | Not at all <input type="checkbox"/> | |
| 3. Over the last week, how much has your skin interfered with you going shopping or looking after your home or garden ? | Very much <input type="checkbox"/> | |
| | A lot <input type="checkbox"/> | |
| | A little <input type="checkbox"/> | |
| | Not at all <input type="checkbox"/> | Not relevant <input type="checkbox"/> |
| 4. Over the last week, how much has your skin influenced the clothes you wear? | Very much <input type="checkbox"/> | |
| | A lot <input type="checkbox"/> | |
| | A little <input type="checkbox"/> | |
| | Not at all <input type="checkbox"/> | Not relevant <input type="checkbox"/> |
| 5. Over the last week, how much has your skin affected any social or leisure activities? | Very much <input type="checkbox"/> | |
| | A lot <input type="checkbox"/> | |
| | A little <input type="checkbox"/> | |
| | Not at all <input type="checkbox"/> | Not relevant <input type="checkbox"/> |
| 6. Over the last week, how much has your skin made it difficult for you to do any sport ? | Very much <input type="checkbox"/> | |
| | A lot <input type="checkbox"/> | |
| | A little <input type="checkbox"/> | |
| | Not at all <input type="checkbox"/> | Not relevant <input type="checkbox"/> |
| 7. Over the last week, has your skin prevented you from working or studying ? | Yes <input type="checkbox"/> | |
| | No <input type="checkbox"/> | Not relevant <input type="checkbox"/> |
| If "No", over the last week how much has your skin been a problem at work or studying ? | A lot <input type="checkbox"/> | |
| | A little <input type="checkbox"/> | |
| | Not at all <input type="checkbox"/> | |
| 8. Over the last week, how much has your skin created problems with your partner or any of your close friends or relatives ? | Very much <input type="checkbox"/> | |
| | A lot <input type="checkbox"/> | |
| | A little <input type="checkbox"/> | |
| | Not at all <input type="checkbox"/> | Not relevant <input type="checkbox"/> |
| 9. Over the last week, how much has your skin caused any sexual difficulties ? | Very much <input type="checkbox"/> | |
| | A lot <input type="checkbox"/> | |
| | A little <input type="checkbox"/> | |
| | Not at all <input type="checkbox"/> | Not relevant <input type="checkbox"/> |
| 10. Over the last week, how much of a problem has the treatment for your skin been, for example by making your home messy, or by taking up time? | Very much <input type="checkbox"/> | |
| | A lot <input type="checkbox"/> | |
| | A little <input type="checkbox"/> | |
| | Not at all <input type="checkbox"/> | Not relevant <input type="checkbox"/> |

Please check you have answered EVERY question. Thank you.

DERMATOLOGY LIFE QUALITY INDEX (DLQI) - INSTRUCTIONS FOR USE

The Dermatology Life Quality Index questionnaire is designed for use in adults, i.e. patients over the age of 16. It is self explanatory and can be simply handed to the patient who is asked to fill it in without the need for detailed explanation. It is usually completed in one or two minutes.

SCORING

The scoring of each question is as follows:

Very much	scored 3
A lot	scored 2
A little	scored 1
Not at all	scored 0
Not relevant	scored 0
Question 7, prevented work or studying	scored 3

The DLQI is calculated by summing the score of each question resulting in a maximum of 30 and a minimum of 0. The higher the score, the more quality of life is impaired.

HOW TO INTERPRET MEANING OF DLQI SCORES

0	1	no effect at all on patient's life
2	5	small effect on patient's life
6	10	moderate effect on patient's life
11	20	very large effect on patient's life
21	30	extremely large effect on patient's life

REFERENCES

Finlay AY and Khan GK. Dermatology Life Quality Index (DLQI): a simple practical measure for routine clinical use. *Clin Exp Dermatol* 1994; **19**:210-216.

Basra MK, Fenech R, Gatt RM, Salek MS and Finlay AY. The Dermatology Life Quality Index 1994-2007: a comprehensive review of validation data and clinical results. *Br J Dermatol* 2008; **159**:997-1035.

Hongbo Y, Thomas CL, Harrison MA, Salek MS and Finlay AY. Translating the science of quality of life into practice: What do dermatology life quality index scores mean? *J Invest Dermatol* 2005; **125**:659-64.

There is more information about the DLQI, including over 85 translations, at www.dermatology.org.uk. The DLQI is copyright but may be used without seeking permission by clinicians for routine clinical purposes. For other purposes, please contact the copyright owners.

name	age	sex	occupation	marital	education	religion	socioeco status	IP /OP	substance use	family psy h/o	family support	psoriasis type	past psy h/o	PASI score	DQI	HAM D	HAM A
s	45	M	coolie	married	primary	hindu	low	OP	-	nil	good	plaque	nil	6	6	5	10
s	56	F	farmer	widow	nil	hindu	low	OP	-	nil	good	plaque	nil	4	14	17	8
f	34	M	driver	married	nil	hindu	low	OP	alcohol	nil	good	plaque	nil	5	9	5	7
g	35	M	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	4	8	4	9
a	45	M	coolie	married	primary	hindu	low	OP	nicotine	nil	good	plaque	nil	5	7	6	7
g	57	F	nil	widow	nil	hindu	low	OP	-	nil	good	plaque	nil	6	10	7	11
s	66	F	coolie	married	primary	hindu	low	IP	-	nil	good	plaque	nil	5	12	24	18
j	34	F	nil	married	nil	hindu	low	OP	-	nil	good	plaque	nil	14	23	16	12
h	35	F	housewife	married	nil	hindu	low	OP	-	nil	good	plaque	nil	5	14	3	10
f	44	F	nil	married	primary	hindu	low	OP	-	nil	good	plaque	nil	4	18	6	9
d	54	F	nil	married	nil	hindu	low	OP	-	nil	good	plaque	nil	3	16	4	8
g	53	F	housewife	married	primary	christian	low	OP	-	nil	good	plaque	nil	5	9	19	17
g	43	M	nil	married	nil	hindu	low	OP	-	nil	good	plaque	nil	5	25	18	11
f	45	M	coolie	married	nil	hindu	low	OP	nicotine	nil	good	plaque	nil	6	16	5	12
d	46	M	farmer	married	nil	hindu	low	OP	-	nil	good	plaque	nil	5	6	3	9
s	44	M	nil	married	nil	christian	low	IP	-	nil	good	plaque	nil	14	18	21	8
a	53	M	coolie	married	primary	hindu	low	OP	alcohol	nil	good	scalp	nil	5	8	16	21
r	45	M	driver	married	primary	hindu	low	OP	-	nil	good	plaque	nil	10	14	3	11
g	32	F	housewife	Unmarried	nil	hindu	low	IP	-	nil	good	plaque	nil	12	24	21	10
d	53	F	housewife	married	primary	muslim	low	IP	-	nil	poor	plaque	depression	13	21	19	22
e	45	F	farmer	married	primary	christian	low	OP	-	nil	good	plaque	nil	4	6	17	5
a	46	F	nil	married	nil	christian	low	OP	-	nil	good	plaque	nil	4	8	2	4
f	55	F	housewife	married	primary	hindu	low	OP	-	nil	good	plaque	nil	5	5	6	13
r	44	F	sales	married	degree	muslim	low	OP	-	nil	good	plaque	nil	6	4	5	4
d	28	F	student	married	bsc	hindu	low	OP	-	nil	good	plaque	nil	6	3	12	7
g	51	M	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	5	6	4	5
t	49	M	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	4	4	16	9
f	33	M	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	3	8	4	6
a	35	M	fisherman	married	nil	hindu	low	OP	-	nil	good	plaque	nil	4	12	4	7
s	35	F	nil	Unmarried	nil	hindu	low	OP	-	nil	good	plaque	nil	5	14	15	21
a	28	M	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	5	9	6	6
d	52	F	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	4	20	16	16
f	57	F	housewife	married	nil	hindu	low	OP	-	nil	good	plaque	nil	5	16	5	7
r	29	M	student	Unmarried	college	hindu	low	OP	-	nil	poor	scalp	nil	7	14	10	21
d	55	M	coolie	married	nil	hindu	low	OP	alcohol	nil	good	plaque	nil	5	12	4	6
s	25	M	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	4	9	18	12
e	33	F	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	4	5	4	6
s	58	F	coolie	married	nil	hindu	low	OP	-	nil	poor	plaque	nil	4	4	6	6
j	32	M	coolie	married	primary	hindu	low	OP	-	nil	good	plaque	nil	2	22	19	13
u	36	M	nil	Unmarried	nil	hindu	low	OP	-	nil	good	plaque	nil	3	19	6	21
h	59	F	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	4	16	4	7
g	54	M	coolie	married	nil	hindu	low	OP	alcohol	nil	poor	plaque	nil	5	12	3	8
d	38	M	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	6	9	22	9
a	44	F	housewife	married	nil	christian	low	OP	-	nil	good	plaque	nil	5	15	6	7
d	39	M	engineer	married	degree	hindu	low	OP	alcohol	nil	good	plaque	nil	6	14	7	8
s	24	M	driver	married	nil	hindu	low	OP	nicotine	nil	good	plaque	nil	5	21	22	16
e	39	F	housewife	married	primary	hindu	low	OP	-	nil	poor	plaque	nil	7	10	2	8
d	42	M	farmer	married	nil	hindu	low	OP	-	nil	good	plaque	nil	5	8	5	9
r	41	M	coolie	married	nil	hindu	low	OP	alcohol	nil	good	plaque	nil	5	9	3	6
f	39	F	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	4	12	4	5
d	35	M	nil	married	nil	muslim	low	OP	nicotine	nil	good	plaque	nil	5	14	22	21
s	28	M	coolie	Unmarried	nil	hindu	low	OP	-	nil	good	plaque	nil	5	16	13	4
a	45	M	auto driver	married	primary	hindu	low	IP	-	nil	poor	plaque	nil	21	10	7	9
s	43	M	nil	married	nil	hindu	low	OP	nicotine	nil	good	plaque	nil	5	14	26	11
a	47	F	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	5	16	7	15
x	46	F	housewife	married	primary	hindu	low	OP	-	nil	good	plaque	nil	12	18	24	12
a	38	F	housewife	married	nil	hindu	low	OP	-	nil	good	plaque	nil	4	26	12	9
c	36	F	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	6	6	4	5
a	46	F	housewife	married	nil	muslim	low	IP	-	nil	good	plaque	nil	17	22	17	18
b	43	M	coolie	married	nil	hindu	low	OP	nicotine	nil	good	plaque	nil	6	9	5	5
b	24	F	coolie	married	primary	hindu	low	OP	-	nil	good	plaque	nil	16	16	15	21
z	37	M	coolie	married	primary	hindu	low	OP	alcohol	nil	poor	plaque	nil	5	23	22	7
a	28	M	coolie	Unmarried	nil	muslim	low	OP	nicotine	nil	good	plaque	nil	4	5	6	8
r	33	M	nil	married	nil	hindu	low	IP	alcohol	nil	good	scalp	nil	16	22	23	22
e	46	F	tailor	married	nil	hindu	low	OP	-	nil	good	plaque	nil	4	9	18	7
d	44	F	housewife	married	nil	muslim	low	OP	-	nil	good	plaque	nil	5	8	4	6
f	52	F	housewife	married	nil	hindu	low	OP	-	nil	good	plaque	nil	4	7	18	5
t	42	F	housewife	married	nil	hindu	low	OP	-	nil	good	plaque	nil	4	6	7	8
s	53	F	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	5	6	12	6
a	32	M	tailor	married	primary	hindu	low	OP	nicotine	nil	poor	plaque	nil	14	21	7	21
s	31	M	nil	married	nil	hindu	low	OP	nicotine	nil	good	plaque	nil	5	8	17	8
b	49	F	housewife	married	nil	christian	low	OP	-	nil	good	plaque	nil	4	5	7	9
u	38	M	nil	married	primary	hindu	low	OP	alcohol	nil	good	plaque	nil	15	21	14	21
h	62	F	nil	Unmarried	primary	muslim	low	OP	-	nil	good	plaque	nil	6	5	7	6
g	47	F	farmer	married	primary	hindu	low	OP	-	nil	good	plaque	nil	7	4	7	8
k	48	M	coolie	married	nil	hindu	low	OP	alcohol	nil	good	plaque	nil	5	6	6	7
a	56	F	housewife	married	nil	hindu	low	OP	-	nil	good	plaque	nil	7	20	17	6
j	27	M	nil	Unmarried	nil	christian	low	IP	alcohol	nil	good	plaque	nil	5	20	15	7
h	29	F	coolie	Unmarried	nil	christian	low	OP	-	nil	good	plaque	nil	7	5	4	6
t	49	F	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	6	6	7	9
f	48	F	coolie	married	nil	hindu	low	IP	-	nil	good	plaque	nil	12	20	19	8
v	56	F	coolie	married	nil	hindu	low	IP	-	nil	good	scalp	nil	16	11	8	21
s	57	F	farmer	married	nil	christian	low	OP	-	nil	good	plaque	nil	5	12	6	8
d	45	M	nil	married	nil	hindu	low	OP	-	nil	good	plaque	nil	15	21	21	9
s	44	M	tailor	married	degree	hindu	low	OP	alcohol	nil	good	plaque	nil	6	8	7	11
a	46	M	farmer	married	nil	hindu	low	OP	-	nil	good	plaque	nil	5	5	7	6
a	52	F	tailor	married	primary	hindu	low	OP	-	nil	good	plaque	nil	4	18	14	12
e	61	F	housewife	married	nil	muslim	low	OP	-	nil	good	plaque	nil	5	4	7	10
c	42	M	farmer	married	nil	muslim	low	IP	alcohol	nil	good	guttate	nil	14	22	25	21
r	37	F	tailor	married	degree	christian	low	OP	-	nil	good	plaque	nil	5	3	6	5
a	33	M	coolie	married	nil	hindu	low	OP	nicotine	nil	good	plaque	nil	6	5	5	7
a	28	M	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	6	6	7	8
l	64	M	nil	widower	nil	hindu	low	IP	alcohol	nil	good	plaque	nil	14	23	18	21
o	59	F	tailor	married	degree	hindu	low	OP	-	nil	good	plaque	nil	5	6	13	7
j	36	F	housewife	married	primary	hindu	low	OP	-	nil	good	plaque	nil	6	9	7	5
a	55	F	housewife	married	nil	christian	low	OP	-	nil	good	plaque	nil	5	6	6	7
h	33	M	coolie	married	nil	christian	low	OP	nicotine	nil	good	plaque	nil	4	5	7	8
g	38	F	farmer	married	nil	christian	low	OP	-	nil	good	plaque	nil	6	8	5	8
a	33	F	housewife	married	nil	christian	low	OP	nicotine	nil	good	plaque	nil	5	7	6	9
a	29	M	coolie	married	nil	hindu	low	OP	-	nil	good	plaque	nil	16	8	17	22